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THE OCCIPITO-ATLANTO-AXIAL JOINTS IN RHEUMATOID ARTHRITIS AND ANKYLOSING SPONDYLITIS*

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THE occipito-atlanto-axial region has special significance in "rheumatoid disease"* because it contains synovial tissue and important ligaments closely arranged within a small, critical space. Nontraumatic, atlanto-axial dislocation has been reported in ankylosing spondylitis and also in rheumatoid arthritis,[3,6,14,17,18,21,22,32] but the prevalence and significance of anterior displacement of the atlas in these conditions have not been generally appreciated. At times, anterior dislocation of the atlas was the initial manifestation of rheumatoid disease[31,44] and occasionally its relationship to coexisting chronic arthritis was apparently not recognized.[33,38] Furthermore, only a few reports have emphasized that the cervical spine may be affected not only in children but also in adults with classic rheumatoid arthritis.[5,10,22,37]

Neck pain and stiffness are not uncommon in rheumatoid disease. In view of the frequency of peripheral joint subluxations in such patients, it was of interest to obtain flexion views of the cervical spine to learn whether these joints were similarly affected. Subluxations, especially atlanto-axial and vertebral erosions, particularly of the odontoid process, were common. A vertical displacement of the cervical spine with respect to the foramen magnum was a frequent finding.

ANATOMY

The odontoid process forms median synovial joints anteriorly with the atlas and posteriorly with the transverse ligament (Fig. 1). This ligament is responsible for the stability of the atlanto-odontoid joint and prevents the atlas from slipping forward when the head is flexed (Fig. 2). It may contain a cartilaginous body against which the dens articulates, and a synovial bursa may be found behind it. The atlanto-axial joints function chiefly in rotation but

* The term "rheumatoid disease" is used here in a general sense to include rheumatoid arthritis, ankylosing spondylitis and the so-called rheumatoid variants, such as juvenile rheumatoid arthritis and arthritis associated with psoriasis or with chronic idiopathic ulcerative colitis.

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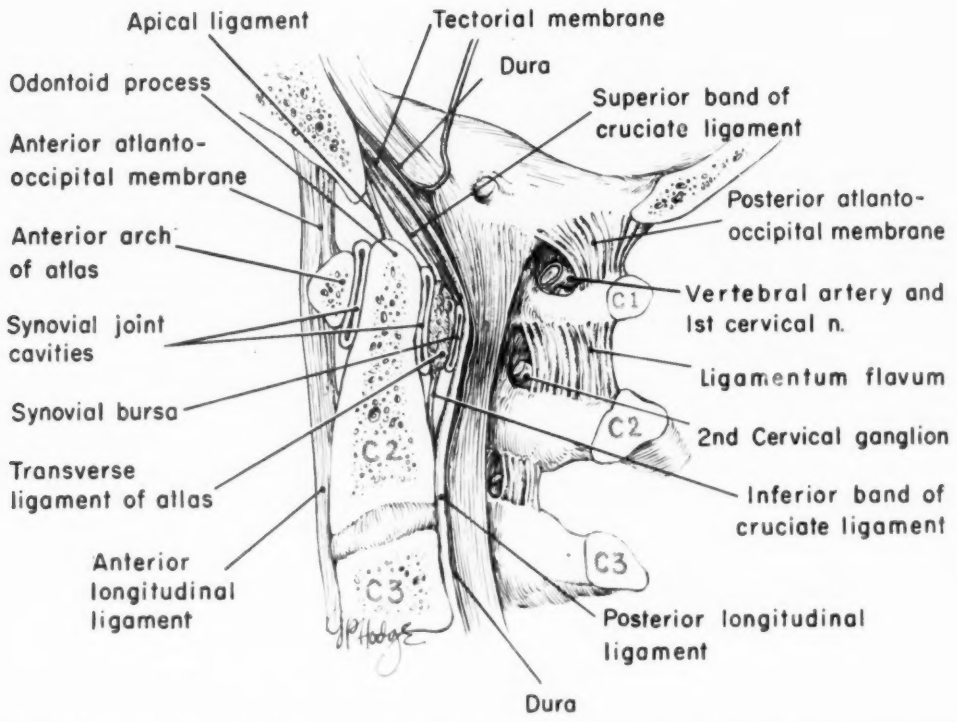

FIG. 1. Mid-sagittal section. Relationship of synovial tissue and ligaments to the atlas, odontoid and foramen magnum. An inconstant synovial bursa may also be found beneath the anterior margin of the foramen magnum.

normally also provide a minimal degree of flexion-extension motion. Thus, a cranial shift of the anterior atlas relative to the dens can be noted in extension and a slight gaping of the superior aspect of the atlanto-odontoid space may be seen in flexion.[44] For practical purposes, this space is constant in adults in flexion and extension, especially inferiorly, and is said not to exceed 2.5 mm.[16] It is apparently impossible to separate the odontoid from the atlas in cadavers unless the transverse ligament is completely cut.[44] In such instances the alar ligaments[44] and possibly also the external occipito-atlanto-axial ligaments[27] limit the degree of forward displacement of the atlas and skull.

The tectorial membrane and the external cranio-vertebral ligaments have been regarded as limiting the range of flexion-extension of the head, a motion which occurs chiefly in the atlanto-occipital joints. Werne[44] believes that flexion in these joints may normally be checked by skeletal contact between the anterior margin of the foramen magnum and the apex of the dens via an interposed synovial bursa. Trolard[42] also noted an inconstant supra-odontoid bursa beneath the anterior margin of the foramen magnum. An anomalous median "third condyle" on the inferior aspect of the basilar process, sometimes bearing an articular facet, has been described.[30] This may represent a "manifestation of an occipital vertebra."[4]

Basilar invagination may be congenital or acquired and implies an upturning of the foramen magnum margins associated with a vertical shift of the spine with respect to the skull. This condition is suggested when the odontoid tip lies more than 4.5 mm. above McGregor's base line, from the hard palate to the inferior margin of the occipital curve.[26] Another, more direct index of basilar invagination is the angle subtended by the clivus and plane of the foramen

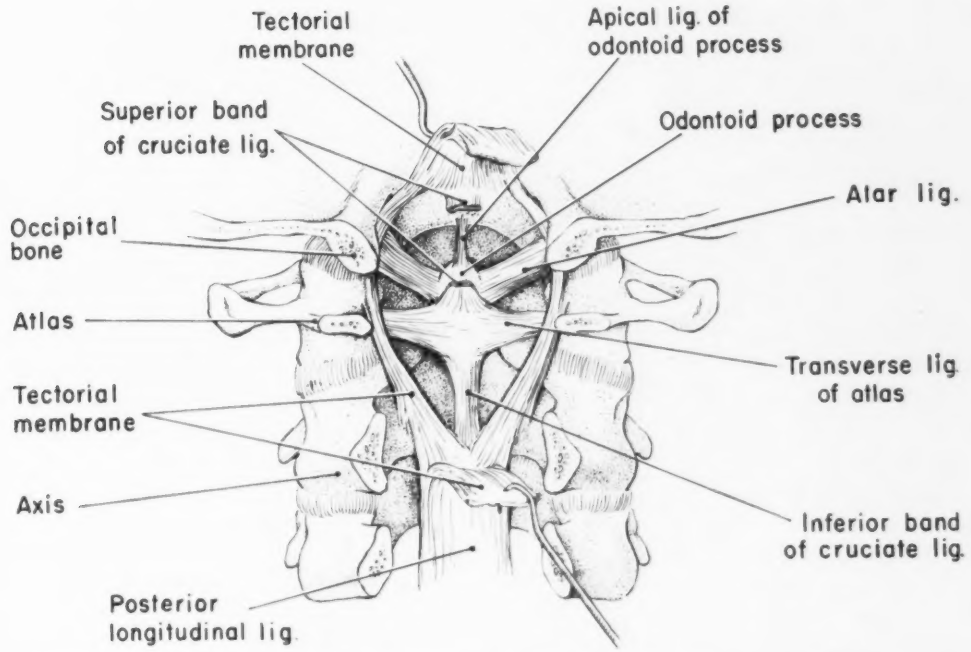

FIG. 2. Coronal section. Posterior view. The transverse ligament keeps the odontoid closely applied to the anterior atlas. (Synovial tissue and vertebral arteries are not shown.)

magnum which normally is said not to exceed 136°.[2,28]

METHODS AND MATERIAL

Lateral roentgenograms of the cervical spine were obtained in flexion and extension in 34 patients with rheumatoid arthritis and 32 with ankylosing spondylitis who were selected because of persistent neck discomfort or severe, progressive disease. Lateral midline laminagrams of the cervical spine were made in 60 of these patients and in 20 cases either open-mouth views of the odontoid process or frontal laminagrams were also obtained. The lumbosacral spine was examined in all but one patient with rheumatoid arthritis. Almost all with rheumatoid arthritis and approximately one-third of those with ankylosing spondylitis were current patients at the University Hospital. The others had been examined during the last four years and were recalled specifically for this study because neck discomfort had been a significant symptom. Three patients with rheumatoid arthritis and one with ankylosing spondylitis were included because a cervical subluxation was clinically suspected or known to have been present. Ankylosing spondylitis was differentiated from rheumatoid arthritis on clinical grounds[8,12] and on the basis of characteristic roentgen findings in the lower spine and pelvis in this disease.[34]

RESULTS*

The clinical data are summarized in Table 1. None of these patients were less than nineteen years of age, although 2 had the onset of rheumatoid arthritis at ages two and fourteen, and one had ankylosing spondylitis at the age of fifteen.

In these patients with rheumatoid arthritis, the disease was of long duration with persistently active synovitis and progressive joint deformities unappreciably altered by any therapeutic measures. Many had excessive and sometimes spectacular periarticular bone resorption, widespread rheumatoid nodule formation and strongly positive tests for the rheumatoid factor.

* Some of these cases have been previously reported.[22]

TABLE I

CLINICAL DATA

	Rheumatoid Arthritis	Ankylosing Spondylitis
Males	8	25
Females	26	7
Totals	34	32
Age (yr.)		
<30	3	3
30–39	3	3
40–49	9	16
50–59	13	6
>59	6	4
Duration (yr.)		
<10	10	3
10–20	14	14
>20	10	15
Rheumatoid Nodules	17	0
Rheumatoid Factor		
No. positive	21	4
No. tested	30	13
Steroid Therapy		
3 mo.–1 yr.	8	2
>1 yr.	23	3
Peripheral Joint Classification*		
Stage I	0	0
Stage II	2	0
Stage III	23	2
Stage IV	9	4
	34	6

* American Rheumatism Association criteria.[40]

Neck discomfort was or had been present in most cases, but in some instances of severe cervical instability this was not a prominent symptom. Subluxations of the peripheral joints were common and at least 7 patients had spontaneous tendon ruptures. The disease was also severe in the majority of patients with ankylosing spondylitis and many had virtually complete spinal rigidity. Almost all had neck discomfort and 14 patients received roentgen therapy to the cervical spine. Eleven of the 32 patients had hip or shoulder involvement and in 6 others the peripheral joints were affected as well.

The cervical spine roentgen findings are summarized in Table II.* Erosions of the odontoid cortex were present in approximately one-third of the patients with rheumatoid arthritis, but were less frequently demonstrated in ankylosing spondylitis. Bony ankylosis of the odontoid to the anterior atlas or basilar process, present in at least 7 patients, may have precluded the demonstration of such erosions in the latter group. There was an associated reactive sclerosis of the odontoid ("shining odontoid") in 3 of the 4 patients with ankylosing spondylitis who exhibited such surface erosions. Reactive sclerosis of the odontoid was apparent in one other patient with spondylitis and in one case of rheumatoid arthritis; in the latter the body of the axis was also sclerotic and eroded. There were cervical vertebral erosions other than in the odontoid in 9 patients with classic

TABLE II

CERVICAL SPINE ROENTGENOGRAPHIC FINDINGS IN 66 ADULTS

	Rheumatoid Arthritis	Ankylosing Spondylitis
Patients	34	32
Odontoid Erosions	12	4
"Shining Odontoid"	1(?)	4
Atlanto-Odontoid Distance (normal limit, 2.5 mm.)		
3–5 mm.	13	5
>5 mm.	11	0
Other Cervical Subluxations	10	4
Baseline-Odontoid Distance (normal limit, 4.5 mm.)		
5–8 mm.	5	7
>8 mm.	5	2
Foramen Magnum-Clivus Angle 136° or Greater	4	1

* Measurements given are uncorrected for roentgenographic enlargement. The correction factor varied (83–87 per cent), depending on the spine-film distance. The target-film distance was 60 inches for conventional roentgenograms and 58 inches for laminagrams.

rheumatoid arthritis. These were located in the vertebral end plates in 4 instances and at the tips of the spinous processes, usually C6, C7 or T1, in 8. Of these 9 patients, 4 had a normal odontoid process.

Abnormal atlanto-odontoid separation was present in 29 patients and, of these, flexion views were required for the diagnosis in 23. Of the 11 patients in which this was severe (greater than 5 mm.), 8 had no other cervical subluxations. Cervical subluxations other than atlanto-axial were frequent and were multiple (exclusive of C1–C2) in 5 patients with rheumatoid arthritis. These figures do not include instances of minimal malalignment. There were no cases of ankylosing spondylitis with multiple cervical subluxations.

Two patients with rheumatoid arthritis exhibited ankylosis of the posterior vertebral joints but neither had paraspinal ossifications and the disease began in childhood in both; one of these had bony ankylosis of the odontoid to the anterior atlas and basilar process. In another patient with rheumatoid arthritis who had severe, immobile anterior subluxation of the atlas of two years' duration, there was a suggestion of calcium deposits in the soft tissues of the atlanto-odontoid space and minimal periosteal new bone formation of the odontoid tip, but no bony ankylosis.

Upward displacement of the spine was common in both conditions. At times this was severe, especially in rheumatoid arthritis, and was associated with a wide foramen magnum-clivus angle with significant frequency.

REPORT OF CASES

RHEUMATOID ARTHRITIS

Case 1 (R. K. No. A2935).† A sixty year old male, who had moderately severe rheumatoid arthritis for twenty-two years, complained of recent neck pain and weakness. He received steroid therapy during the preceding months because of an exacerbation of his peripheral joint manifestations. Generalized lymphadenopathy and severe limitation of neck motion were present. There was a tender, subcutaneous nodule in the occiput. Multiple peripheral joints showed chronic, active synovitis. Neurologic examination was negative except for muscular weakness. The latex fixation test for the rheumatoid factor was strongly positive (1:5120). A roentgenogram showed an anterior subluxation of the atlas in flexion (Fig. 3). The sacroiliac joints were normal. He developed a left hemiplegia and expired. Cerebral arteriography had revealed occlusion of the right internal carotid artery. At necropsy the atlanto-axial subluxation was confirmed and bilateral cerebral infarcts were demonstrated. Microscopic examination of the odontoid process showed erosions of its cartilage and cortex, associated with ingrowth of proliferating pannus (Fig. 4*A*) and an adjacent, chronic, nonspecific synovitis. Ligaments adjacent to the odontoid showed areas of fibrinoid necrosis with chronic inflammation (Fig. 4*B*).

Fig. 3. Case 1. Rheumatoid arthritis. Lateral view in flexion. The atlanto-odontoid distance (arrow) measured 7 mm. (Courtesy of *Arthritis and Rheumatism*.)

Case 11 (D. S. No. 756575). A fifty-four year old male had severe rheumatoid arthritis of multiple peripheral joints since 1948 for which

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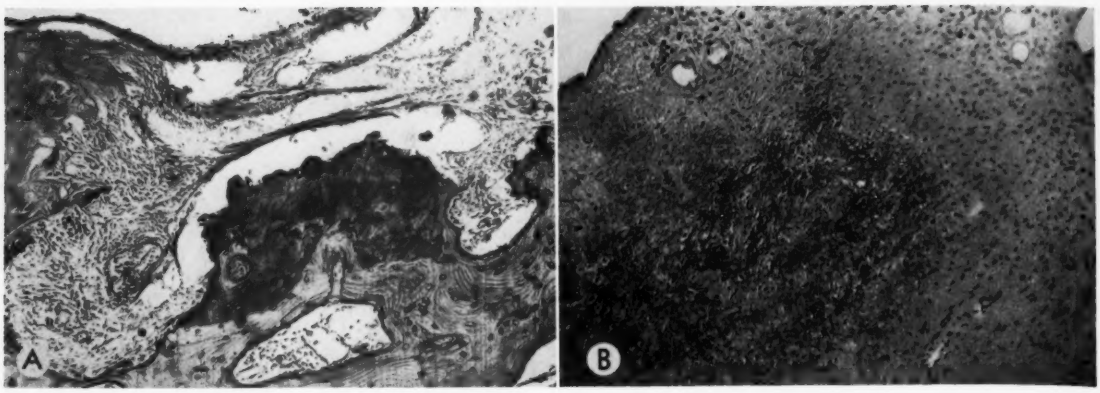

Fig. 4. Case I. Rheumatoid arthritis. (*A*) Odontoid process, ×90. Erosion of cartilage (upper left) and bone, with adjacent pannus. (*B*) Ligament which was adjacent to the odontoid, ×140. Fibrinoid necrosis and chronic inflammation.

he received steroid therapy for a period of eight years. In 1959 he developed neck pain and occipital headaches which were aggravated by head flexion. Neck motion was limited and there was tenderness over the upper cervical spine. He had numerous subcutaneous rheumatoid nodules and multiple extensor digitorum communis tendon ruptures. Neurologic examination was negative. The latex fixation test was positive (1:5120). Cervical roentgenograms and a midline laminagram showed normal vertebral alignment in a neutral position but anterior dislocation of the atlas in flexion; the atlanto-odontoid distance measured 11 mm. The odontoid process was severely eroded and erosions were also present in the vertebral end plates of C6 (Fig. 5, *A*, *B* and *C*).

Case III (J. P. No. 433316). Rheumatoid arthritis began at the age of twenty-one in this

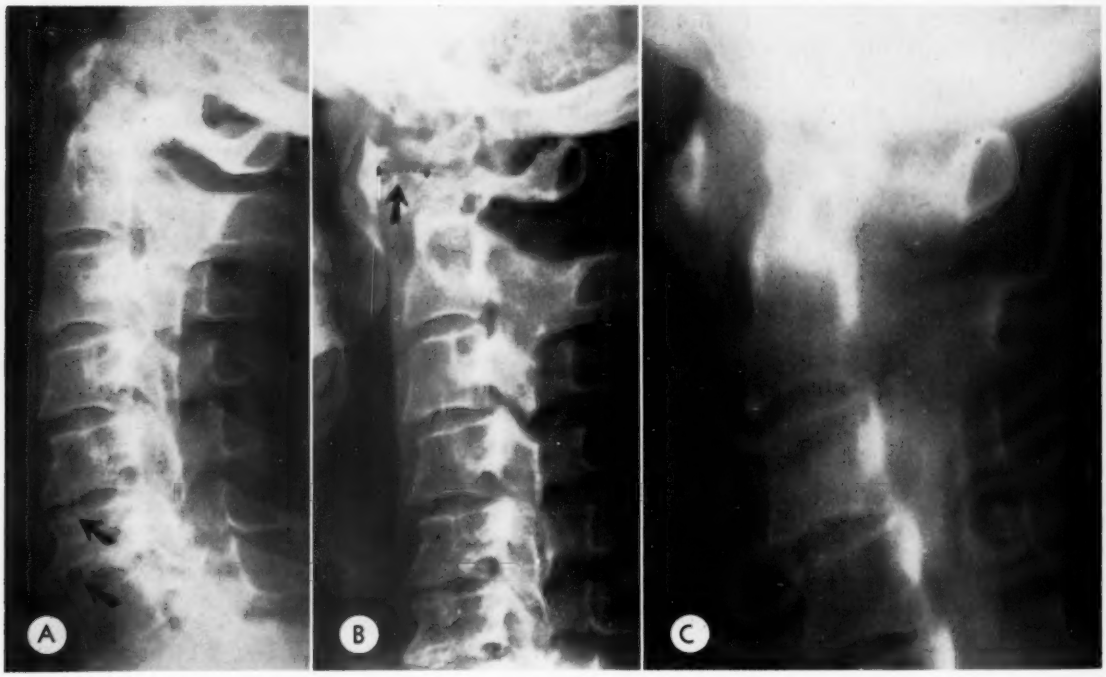

Fig. 5. Case II. Rheumatoid arthritis. (*A*) Extension. Normal atlas alignment. Erosions of C6 (arrows). (*B*) Flexion. Atlanto-odontoid separation of 11 mm. (arrow). (*C*) Midline laminagram. Erosion of the odontoid process. (Courtesy of *Arthritis and Rheumatism*.)

forty-three year old male, but was relatively quiescent until 1958. At that time, after an episode of pneumonia, he developed severe, persistent synovitis of virtually all peripheral joints and was treated with steroids for several weeks. Physical examination in 1960 showed advanced rheumatoid arthritis of many joints and multiple rheumatoid nodules. He had a nasopharyngeal soft tissue mass which proved to be lymphoid hyperplasia. Neurologic findings were negative except for weakness and a gross action tremor which he said began in 1958. The latex fixation test was positive (1:5120). A roentgenogram showed an increase in the atlanto-odontoid distance from 1 to 3 mm. with flexion and an irregular odontoid erosion at the point of articulation with the transverse ligament (Fig. 6).

Case IV (L. B. No. 934431). A nineteen year old male had episodes of migratory polyarthritis at ages fourteen and seventeen. He developed fever, persistent arthritis of several peripheral joints, and neck stiffness two years previously for which he received steroid therapy. Physical examination showed neck motion to be limited. The heart was normal. There was active synovitis in multiple joints. Serologic tests for the rheumatoid factor were negative. Roentgenograms showed bony ankylosis of several cervical apophyseal joints. In flexion the atlanto-odontoid distance increased to 4 mm. He had typical basilar invagination (Fig. 8, *A* and *B*). The odontoid-base line distance was 15 mm. and the foramen magnum-clivus angle was 136°. The sacroiliac joints were normal and there were no paraspinal ossifications.

Fig. 6. Case III. Rheumatoid arthritis. Midline laminagram. Irregular erosion of the posterior surface of the odontoid at the articulation with the transverse ligament. Note nasopharyngeal lymphoid hyperplasia and prominence of retro-odontoid soft tissue.

Fig. 7. Rheumatoid arthritis. Midline laminagram showing an erosion of the posterior aspect of the odontoid with an adjacent bone fragment resembling osteochondritis dissecans. There are multiple cervical subluxations.

Case V (C. S. No. 731351). A forty-two year old female physician had severe, progressive rheumatoid arthritis of virtually all peripheral joints since the age of twenty-four. There was widespread destruction of articular cartilage and bone, especially in the hands and wrists.

Fig. 8. Case iv. Rheumatoid arthritis. (*A*) Lateral view. (*B*) Midline laminagram. Typical basilar invagination and deformity of the vertebral bodies which was associated with ankylosis of the posterior vertebral joints.

She had multiple rheumatoid nodules, lymphadenopathy and splenomegaly. There was no significant limitation of spinal motion. Neurologic findings suggested peripheral neuritis. Serologic tests for the rheumatoid factor were repeatedly positive. A roentgenogram showed multiple cervical subluxations with decrease in the height of the intervertebral disks and erosions of the vertebral end plates, posterior arches, articular processes and odontoid. The latter extended 12 mm. above McGregor's base line and the foramen magnum-clivus angle measured 142°. There was a soft tissue density behind the odontoid which was felt to represent inflammation or brain tissue extending into the spinal canal (Fig. 9).

Case vi (C. T. No. 256613). A fifty-six year old male had severe, progressive rheumatoid arthritis since 1948 with involvement of virtually all peripheral joints. He had marked periarticular bone resorption in the hands and wrists. There were no significant symptoms referable to the spine and no manifestation of spinal cord compression. Serologic tests for the rheumatoid factor were persistently positive. Roentgenograms showed multiple cervical subluxations with erosions of the posterior arches and articular processes. The axis was also eroded and sclerotic and the atlas had descended to its inferior margin; the odontoid process was sclerotic and extended into the posterior fossa. The foramen magnum-clivus angle measured 136° but the upturning of the foramen magnum margins, typical of severe basilar invagination, was absent (Fig. 10, *A* and *B*).

ankylosing spondylitis

Case vii (J. G. No. 528883). A thirty-one year old male had the onset of arthritis of the sacroiliac joints, hips and knees at the age of fifteen. Spinal and costovertebral joint involvement was progressive with severe limitation of motion and reduction in chest expansion. Pain due to cervical spondylitis (Fig. 11*A*) necessitated roentgen therapy in 1954 with some relief. He was unable to rotate his head on physical examination in 1960. Roentgenograms showed that the atlas had become dislocated anteriorly 5 mm. A laminagram demonstrated bony proliferation of the odontoid with fusion to the atlas in the dislocated position (Fig. 11*B*). He also developed an erosion of the spine of C7 during this six year interval. There was complete bony ankylosis of the sacroiliac and posterior vertebral joints with paraspinal ossifications, characteristic of ankylosing spondylitis.

Case viii (N. R. No. 327511). A sixty-one

year old female developed ankylosing spondylitis in 1943. Her spine became progressively rigid and chest expansion was reduced. The peripheral joints were normal. She complained of neck pain (Fig. 12*A*) and stiffness for several years for which she received roentgen therapy (250 r in 1945) and she also noted a cracking sensation on head motion. Roentgenograms and a midline laminagram (Fig. 12*B*) in 1960 showed superficial erosions of the odontoid process, associated with a reactive sclerosis and mottling of the trabecular pattern. The sacroiliac joints and lumbar spine showed characteristic changes of ankylosing spondylitis.

Case ix (E. S. No. 713526). Ankylosing spondylitis and psoriasis developed at the age of thirty-four in this forty-six year old male. His spine became almost totally rigid and chest expansion was severely limited. He had arthritis of almost all peripheral joints. There were no subcutaneous nodules and the serologic tests for the rheumatoid factor were repeatedly negative. Neurologic examination was difficult because of extensive psoriasis and widespread ankylosis of the peripheral joints. Comparative roentgenograms made in 1952 and 1959 showed that the anterior arch of the atlas had become reduced in thickness and that the odontoid had also become small and pointed (Fig. 14, *A* and *B*). These structures were displaced above McGregor's line but were considerably anterior to the foramen magnum, indicating that an atlanto-occipital subluxation had occurred. This was apparently followed by spontaneous atlanto-occipital fusion. There was bony ankylosis of the sacroiliac and virtually all of the posterior vertebral joints and extensive ossifications were present along the paravertebral ligaments.

Case x (L. R. No. 468244). A forty-nine year old male had the onset of spondylitis in 1935. Neck pain has been prominent, necessitating several courses of roentgen therapy (4,150 r between 1940 and 1947). He has had virtually complete spinal rigidity since 1950. The peripheral joints were never involved but temporomandibular joint motion was reduced. Comparative roentgenograms made in 1943 and 1960 showed atrophy of the odontoid and atlas with bony ankylosis of these structures to each other and to the basilar process (Fig. 15, *A* and *B*). He had erosions of the spinous processes of C6 and C7, calcification with reduction in the height of the cervical intervertebral disks and extensive bony ankylosis of the sacroiliac and posterior vertebral joints with paraspinal ossifications, characteristic of ankylosing spondylitis.

Fig. 9. Case v. Rheumatoid arthritis. Midline laminagram. "Stepladder" subluxations with reduction in the height of the intervertebral disks and erosions of the vertebral end-plates, posterior arches and odontoid process. The latter extends well above the palato-occipital line and the foramen magnum-clivus angle is 142°. The lower spinous processes appear "pointed" as does the basilar process. Note retro-odontoid soft tissue mass.

Discussion

The cervical spine is frequently affected in classic rheumatoid arthritis as well as in ankylosing spondylitis. Subluxations, especially atlanto-axial, often accompany such involvement but flexion views are generally required for the diagnosis. Pathologic laxity of the transverse ligament may account for minimal degrees of atlanto-odontoid separation. When this separation is severe, however, one may postulate

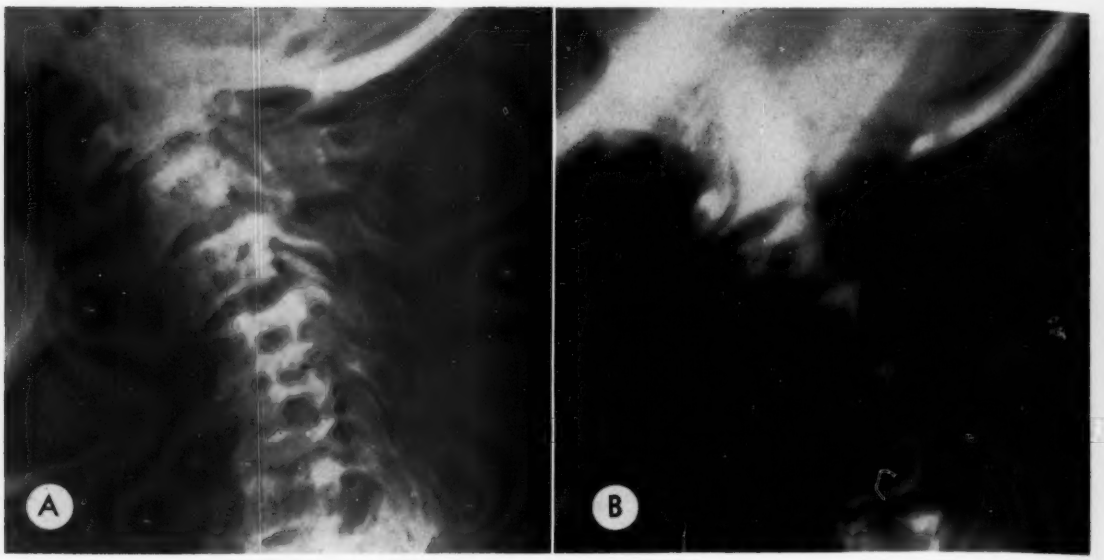

Fig. 10. Case VI. Rheumatoid arthritis. (*A*) Lateral view. Multiple cervical subluxations with severe erosions of the posterior arches and vertebral end-plates. The atlas has descended to the inferior margin of C2. (*B*) Midline laminagram. The body of C2 is sclerotic and eroded. The odontoid too is sclerotic and extends into the posterior fossa. The foramen magnum-clivus angle appears wide but the tip of the basilar process, as well as the occipital condyles and lateral masses have probably been eroded ("pseudo-basilar invagination").

rupture of this ligament; in such cases, the extent of forward dislocation of the atlas and skull is apparently variable and depends largely on the functional length of the alar ligaments.[44] Minimal widening of the atlanto-odontoid space in flexion, in the presence of odontoid erosion, does not necessarily signify an abnormality of the transverse ligament. The reduced diameter of the odontoid due to erosion may cause it to fit loosely in the ring formed by the anterior atlas and transverse ligament and may alone account for atlanto-odontoid instability. Cervical subluxation was less prevalent in ankylosing spondylitis but many had advanced bony fusion of the posterior articulations and extensive paraspinal ossifications. Such subluxations may occur more frequently during the active phase of this disease. It is possible, too, that neck pain is a less reliable indication of cervical subluxation in ankylosing spondylitis than in rheumatoid arthritis.

The occurrence of erosions of the odontoid process in these diseases is not surprising. In rheumatoid arthritis such lesions, as well as those in adjacent ligaments, are apparently identical to those in the peripheral joints (Fig. 4, *A* and *B*). One patient exhibited an erosion of the posterior aspect of the odontoid with an adjacent bone fragment resembling osteochondritis dissecans (Fig. 7). It is conceivable that the cartilaginous body, often found in the transverse ligament, may undergo osseous metaplasia, but in this case, the size and straight, smooth posterior aspect of the bony fragment suggested that it originated from the odontoid process. The lower cervical spinous processes either showed sharply marginated cortical erosions or, more often, their previously rounded ends became pointed, frequently appearing as though they had been amputated. The vertebral end plate erosions (Fig. 9), with reduction in the height of the adjacent intervertebral disks, at times resembled spinal tuberculosis. These occurred at multiple levels in 2 cases, at C2–C3 in one, and at C5–C6 in another. Thinning of the disks, without adjacent osteophytes or erosions, occurred in the upper half of the cervical spine in several patients in whom the disease began in adulthood.

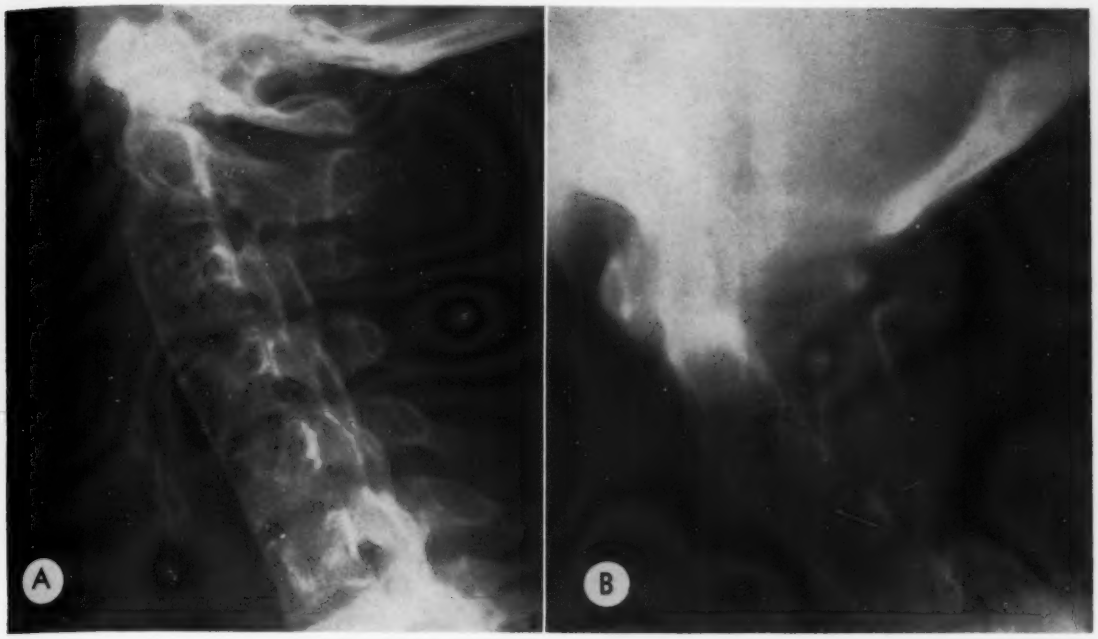

Fig. 11. Case VII. Ankylosing spondylitis. (*A*) 1954. Advanced cervical spondylitis. Normal alignment of the atlas. (*B*) 1960. Midline laminagram showing 5 mm. anterior displacement of the atlas and bone proliferation around the odontoid with fusion to the atlas in the dislocated position.

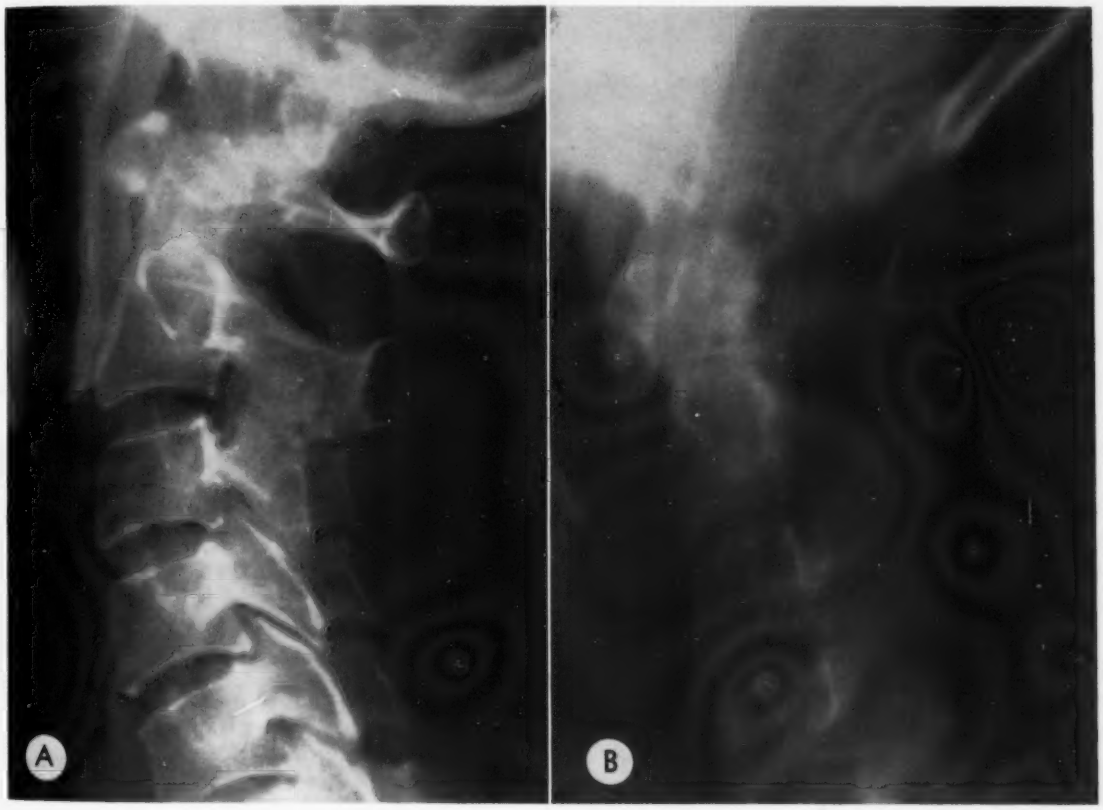

Fig. 12. Case VIII. Ankylosing spondylitis. (*A*) 1945. Normal odontoid. (*B*) 1960. Midline laminagram showing superficial erosions and reactive sclerosis of the odontoid with a mottled trabecular pattern.

Fig. 13. Nineteen year old male. (*A*) Extension. (*B*) Flexion. Minimal atlanto-odontoid separation with flexion. The odontoid is sclerotic with superficial erosions. Neck and back pain were prominent and the sacroiliac joints showed characteristic changes of ankylosing spondylitis.

The surface erosions of the odontoid in ankylosing spondylitis appeared superficial and were usually associated with a reactive sclerosis, obliterating the normal trabecular pattern (Fig. 12, *A* and *B*; and 13). Such erosions with adjacent osteosclerosis have been described in the pelvic bones and vertebral bodies in this disease,[7,13,34] usually at points of ligamentous attachment, and presumably represent the same pathologic process as in the odontoid process. Romanus and Ydén[34] have referred to such lesions in the anterior corners of the vertebral bodies as "shining corners"; hence, the term "shining odontoid" is suggested. The atlas and odontoid became atrophic in 2 ankylosing spondylitis patients who had also developed either atlanto-occipital or atlanto-odontoid-occipital fusion (Case IX and X). These are not listed as "odontoid erosions" in Table II but were, in all probability, due to a subtle erosive process, giving the appearance of a "wearing down of the bone"[34] (Fig. 14 *B* and 15 *B*).

Occasionally, the sacroiliac joints, as well as the cervical spine, are involved in otherwise classic rheumatoid arthritis.[5,36] This appeared to be true in 4 cases in this series in which the sacroiliac joints were narrowed and showed superficial, sharply demarcated marginal irregularities, unattended by reactive sclerosis. Three of the 4 patients also had erosions of the ischial tuberosities. None had paraspinal ossifications (syndesmophytes).[34] These patients differed clinically as well as roentgenologically from those with typical ankylosing spondylitis[23] in that they had rheumatoid nodules and a strongly positive test for the rheumatoid factor, and none had significant limitation of back motion.

It was of interest to correlate the foramen

magnum-clivus angles with the odontoid-baseline distances in these patients and to compare them with values obtained in individuals of similar age distribution, not having rheumatoid disease (Fig. 16). In most cases these were either normal individuals or patients in whom there was a question of a skull fracture. Laminagrams were available in 12 instances. Care was taken to exclude those with proved intracranial pathology or generalized bone disease. There was a statistically significant departure from normal (t test) in the odontoid-baseline distance both in rheumatoid arthritis and ankylosing spondylitis. Upward displacement of the spine relative to the palato-occipital line may be caused by erosions of the cartilage and bone of the lateral atlanto-axial and atlanto-occipital joints and need not be associated with basilar invagination. However, apparently as the ondontoid-baseline distance increases, there is a tendency toward a wide foramen magnum-clivus angle. In rheumatoid arthritis, the foramen magnum-clivus angle differed significantly (t test, $P < .005$) from the control group. There was also a statistically significant difference in ankylosing spondylitis (t test, $.025 < P < .05$) but this was less marked than in rheumatoid arthritis. It is of interest to note that the mean values are not widely divergent (Table III). It appears that secondary basilar invagination can occur as a complication of rheumatoid arthritis and accounts for widening of the foramen magnum-clivus angle and vertical translation of the spine. However, in some patients the up-

TABLE III

MEAN VALUES FOR ODONTOID-BASELINE DISTANCE AND FORAMEN MAGNUM CLIVUS ANGLE

	Odontoid-Baseline Distance (mm.)	Foramen Magnum-Clivus Angle
"Normals"	0.38	121.3°
Rheumatoid Arthritis	3.62	125.6°
Ankylosing Spondylitis	3.03	123.4°

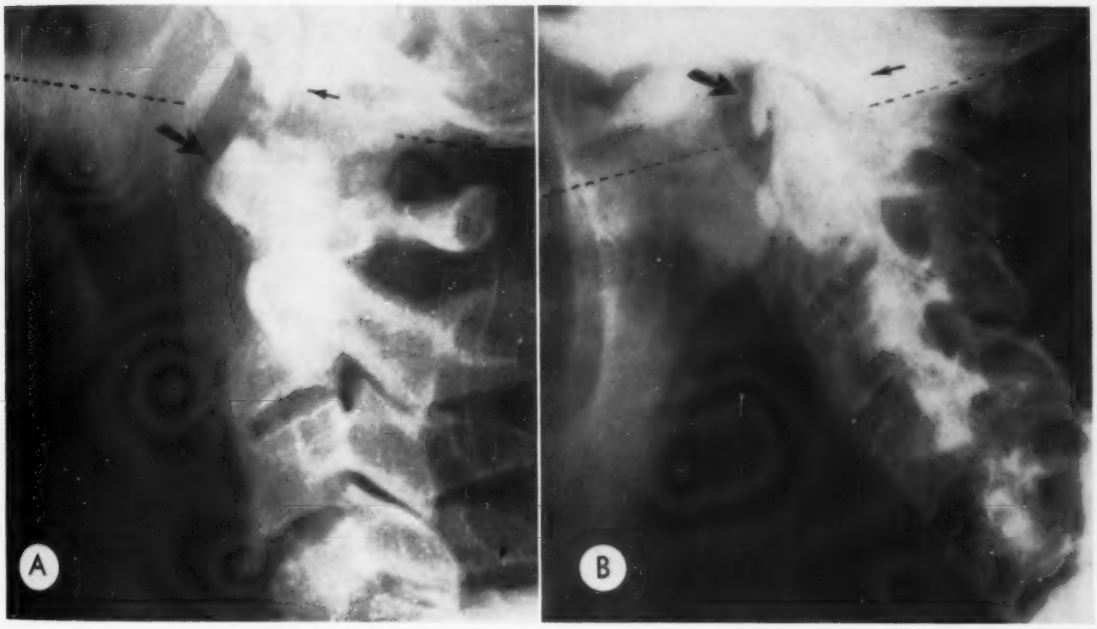

FIG. 14. Case IX. Ankylosing spondylitis. (*A*) 1952. (*B*) 1959. The atlas (large arrow) and odontoid have become atrophic, as though worn down, and displaced upward and anterior to the foramen magnum, indicating atlanto-occipital subluxation. Note tip of basilar process (small arrow) and McGregor's line. Atlanto-occipital fusion apparently followed. No basilar invagination. (Courtesy of *Arthritis and Rheumatism.*)

Fig. 15. Case x. Ankylosing spondylitis. (*A*) 1943. Normal atlas and odontoid. (*B*) Midline laminagram. The atlas (arrow) and odontoid have atrophied and are fused to one another and to the basilar process.

turning of the foramen magnum margins that is typical of basilar invagination is absent and yet the odontoid-baseline distance is markedly abnormal and the foramen magnum-clivus angle is wide. This was true in Case vi where the odontoid extended into the posterior fossa. One may postulate that in some patients not only are the lateral masses and occipital condyles eroded but the tip of the basilar process is likewise destroyed. Such erosions might allow the skull to descend on the spine, as it were, and would also give the appearance of a wide foramen magnum-clivus angle. This situation might properly be differentiated as a "pseudo-basilar invagination."

Laminagrams were indispensable for clearly demonstrating the odontoid lesions and delineating the precise odontoid-atlas relationship. They also showed the periodontoid soft tissues to advantage. In several patients with advanced bony lesions in this region, there was unusual prominence of these soft tissues, possibly due to inflammation. In one patient with basilar invagination, this may have represented brain tissue extending into the spinal canal (Case v).

The lack of significant neurologic manifestations was a perplexing finding. The slow development of these lesions allows an adaptation of the spinal cord, and the reduction in size of the odontoid might explain this discrepancy. However, in several instances the sagittal diameter of the spinal canal was less than the lower normal limits.[45,47]

Spontaneous atlanto-axial subluxation causing quadriplegia has been reported in ankylosing spondylitis,[21] and was also observed in rheumatoid arthritis with fatal spinal cord compression.[22] While muscular atrophy in rheumatoid disease is probably a peripheral effect in most cases, compres-

sion of the spinal cord at the foramen magnum or upper cervical spine may also produce such atrophy and loss of reflexes in the upper extremities. An anterior horn lesion may be due to compression of the anterior spinal artery. It is possible that in some cases significant neurologic manifestations are being overlooked or attributed to the peripheral effects of the disease, for in patients with advanced disease and crip-

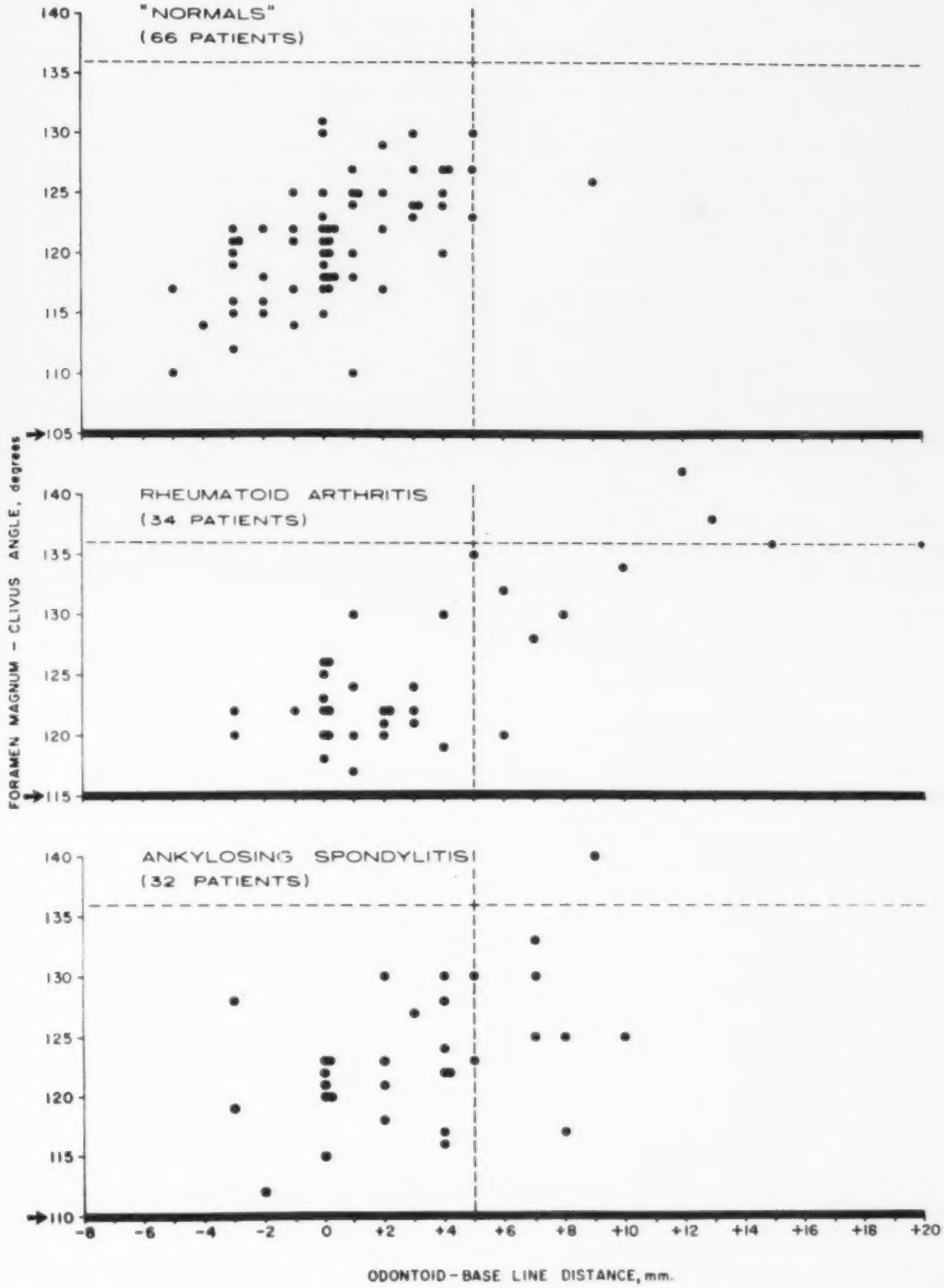

Fig. 16. Correlation of odontoid-baseline distance and foramen magnum-clivus angle in rheumatoid arthritis and ankylosing spondylitis and comparison with "normal" values. Interrupted lines represent the accepted upper normal limits.

pling deformities neurologic examination is often difficult. It is reasonable that chronic spinal cord compression or vertebral artery insufficiency[35] may develop in some instances. The latter may have been a significant factor in Case 1, precluding collateral blood flow in the presence of internal carotid artery occlusion. Reports of delayed myelopathy in traumatic atlanto-axial dislocation[1,43] and in congenital abnormalities around the foramen magnum[20] suggest that a similar asymptomatic interval may obtain in these patients.

Virtually all of the patients with rheumatoid arthritis received steroid therapy, but this may reflect the severity of the disease in these patients rather than a correlation with cervical subluxation. Atlanto-axial subluxation and odontoid erosion have been seen in patients who did not have steroids.[11,22] The prevalence of these potentially dangerous lesions in unselected patients with rheumatoid arthritis remains to be determined, and also whether their development can be correlated with prolonged steroid therapy, age of the patient, duration or severity of the disease, or any other factor.

DIFFERENTIAL DIAGNOSIS

Spontaneous bony fusion in the occipito-atlanto-axial joints of the type seen in ankylosing spondylitis may also occur in rheumatoid arthritis when the disease begins in childhood. This is consistent with the bony ankylosis that can occur in the posterior cervical joints in these cases. In one child with rheumatoid arthritis and chronic atlanto-axial dislocation of two years' duration, laminagrams disclosed periosteal new bone formation of the odontoid process. Children with nonspecific infections of the neck (*e.g.*, tonsillitis, mastoiditis) may develop spontaneous atlanto-axial dislocations.[15,39,41,44,46] This suggests that a specific rheumatoid lesion of the transverse ligament need not necessarily be postulated to explain atlanto-axial subluxation in juvenile rheumatoid arthritis. Congenital malformations,[20,27,29] such as primary basilar invagination, atlas assimilation, and dysplasia of the odontoid, must also be considered in the differential diagnosis. Septic arthritis, pyogenic[9] or tuberculous, rarely involves the upper cervical spine. Cervical subluxations may occur as a complication of degenerative spondylosis and osteoarthritis, but usually involve the lower cervical spine and are generally of minimal degree although at times they may be severe; however, involvement of the atlanto-odontoid joint is not apparently associated with atlanto-odontoid separation. Trauma to the completely ankylosed cervical spine in spondylitis may cause a fracture-dislocation. In these cases the spine fractures like a long bone, with the break usually occurring through what had formerly been the interspace.[19] Neurologic symptoms are probably more apt to occur in these instances than in the spontaneous subluxations in the nonankylosed spine. Multiple cervical subluxations, basilar invagination and severe destruction of the atlas, odontoid and possibly also the basilar process have been observed in lipoid dermatoarthritis in which there was severe involvement of the peripheral joints.[24]

SUMMARY

Rheumatoid arthritis and ankylosing spondylitis often involve the cervical spine, particularly the occipito-atlanto-axial joints. Nontraumatic cervical subluxation is frequent, especially in the atlanto-axial joints. Osseous erosions, particularly of the odontoid process, can often be demonstrated. Such surface erosions of the odontoid process in ankylosing spondylitis are often associated with a sclerosing osteitis. Basilar invagination may occur as a complication of rheumatoid arthritis, but in some cases the features are atypical and might more properly be called "pseudo-basilar invagination." These lesions of the cervical spine are potentially dangerous and often overlooked.

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Vol. 86, No. 2

DESTRUCTIVE LESIONS OF THE VERTEBRAL BODIES IN RHEUMATOID DISEASE*

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DURING recent years there have been occasional reports of destructive lesions of the vertebral bodies occurring in patients with rheumatoid disease.[3,10,19,20,25] These osteopathic lesions of the vertebrae constitute a confusing but perhaps significant feature of the disease.

The first description of a locally destructive lesion involving the upper and lower surfaces of the vertebral bodies was in an article by Andersson[1] on 2 patients with Marie-Strümpell arthritis. Edström[7] in 1940 reported several patients with erosion of the upper and lower corners of the upper lumbar and lower dorsal spine. One patient with a destructive area in the lower anterior portion of the twelfth dorsal vertebra was thought to have a tuberculous spondylitis because of the roentgenographic appearance. This diagnosis was never confirmed and the author concluded that it was a manifestation of rheumatoid spondylitis. Forestier *et al.*[8] described 2 cases with destructive vertebral lesions in a series of 200 cases of Marie-Strümpell arthritis. Roentgenographically, the lesions resembled those of tuberculosis but this disease was never proved. Guest and Jacobson,[10] in discussing osteopathic vertebral lesions occurring in 7 patients with rheumatoid spondylitis, illustrated one with a destructive lesion "involving the adjacent surfaces of the bodies of the eleventh and twelfth thoracic vertebra with peripheral eburnation and disk narrowing (not unlike tuberculous spondylitis)." In 1952, Baggenstoss, Bickel and Ward[3] reported 3 cases of rheumatoid spondylitis with similar destructive lesions of the vertebral bodies. These authors also described a patient with peripheral rheumatoid arthritis and destructive lesions of two vertebral bodies which, at autopsy, proved to be granulomatous, similar histologically to granulomatous nodules found in the synovia, heart and subcutaneous tissues. A more recent report by Wholey, Pugh and Bickel[25] from the same institution described 10 patients with similar roentgenographic lesions.

Romanus and Ydén,[20] after a careful clinical and roentgenographic analysis of 250 cases of rheumatoid spondylitis, concluded that destructive vertebral lesions were not merely sporadic phenomena occurring in occasional cases of rheumatoid spondylitis but could be found in the majority of cases, although they did not state precisely the incidence in their series. According to these authors, the destructive phase is followed relatively soon by a reparative phase that may result in complete healing with no trace of the spondylitic lesions; this may be the reason why it has not been commonly observed. Since roentgenograms are taken at infrequent intervals, these intermediary phases may be undetected.

Romanus and Ydén[20] describe the earliest change as a loss of bony cortex in a small area near the upper and lower anterior corners of the vertebral body which may be accompanied by a varying degree of sclerosis in the adjacent bone. As this progresses, the anterior borders of the vertebral bodies lose their normal convexity and this is responsible for the so-called square vertebrae characteristic of Marie-Strümpell arthritis. These authors feel that only in exceptional cases is this "squaring" due to bone deposition on the central third of the anterior portion of the vertebral body. In several of their patients, such destructive lesions of the vertebral bodies were the earliest roentgenographically demonstrable

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TABLE I

Case No.	Patient	Age at Time of Destructive Lesion	Sex	Type of Arthritis	Duration of Arthritis Preceding Destructive Lesion (yr.)	Site of Destruction	Cortisone Treatment
I	D.L. 147-64-36	44	F	? clinically Marie-Strümpell; roentgenographically Marie-Strümpell	1	Mid-dorsal and upper lumbar	No
II	M.K. 30-94-57	40	M	Marie-Strümpell	17	L_2, L_3	No
III	H.W. 06-67-39	34	F	Marie-Strümpell	7	L_1, L_2	Yes (8 yr.)
IV	I.P. 08-89-67	55	F	Peripheral rheumatoid arthritis	9	T_8, T_9	Yes
V	N.B. 03-75-84	48	F	Peripheral rheumatoid arthritis	11	C_6, C_7	Yes (9 yr.)
VI	F.D. 79-94-59	36	M	Marie-Strümpell	18	T_5, T_6	Yes
VII	H.D. 01-85-79	29	M	Marie-Strümpell	9	L_3, L_4	Yes
VIII	H.E. 11-94-82	53	M	Marie-Strümpell	12	T_7, T_8, T_{11}, T_{12}	Yes (2 yr.)
IX	H.R. 147-48-29	47	M	? clinically Marie-Strümpell; roentgenographically Marie-Strümpell	3	L_2, L_3, L_4	No
X	H.K. 04-76-92	27	M	Marie-Strümpell	10	L_2, L_3	No
XI	S.C. 96-31-33	38	M	Marie-Strümpell	15	T_{10}, T_{11}, T_{12}	Yes
XII	R.A. 17-40-29	39	F	Marie-Strümpell	8	L_2, L_3, L_5	Yes
XIII	M.H. 66-63-75	58	F	Marie-Strümpell	24	T_{10}, T_{11}	No

sign of spinal involvement. Although it always occurred after involvement of the sacroiliac joints, it was frequently a helpful diagnostic sign. There seems to be a predilection for the vertebral bodies at the dorsolumbar and lumbosacral junction.

Our experience during the last few years with several patients who had similar destructive lesions of the vertebral bodies prompted a review of the roentgenograms of 110 patients with Marie-Strümpell arthritis. This survey disclosed 11 patients

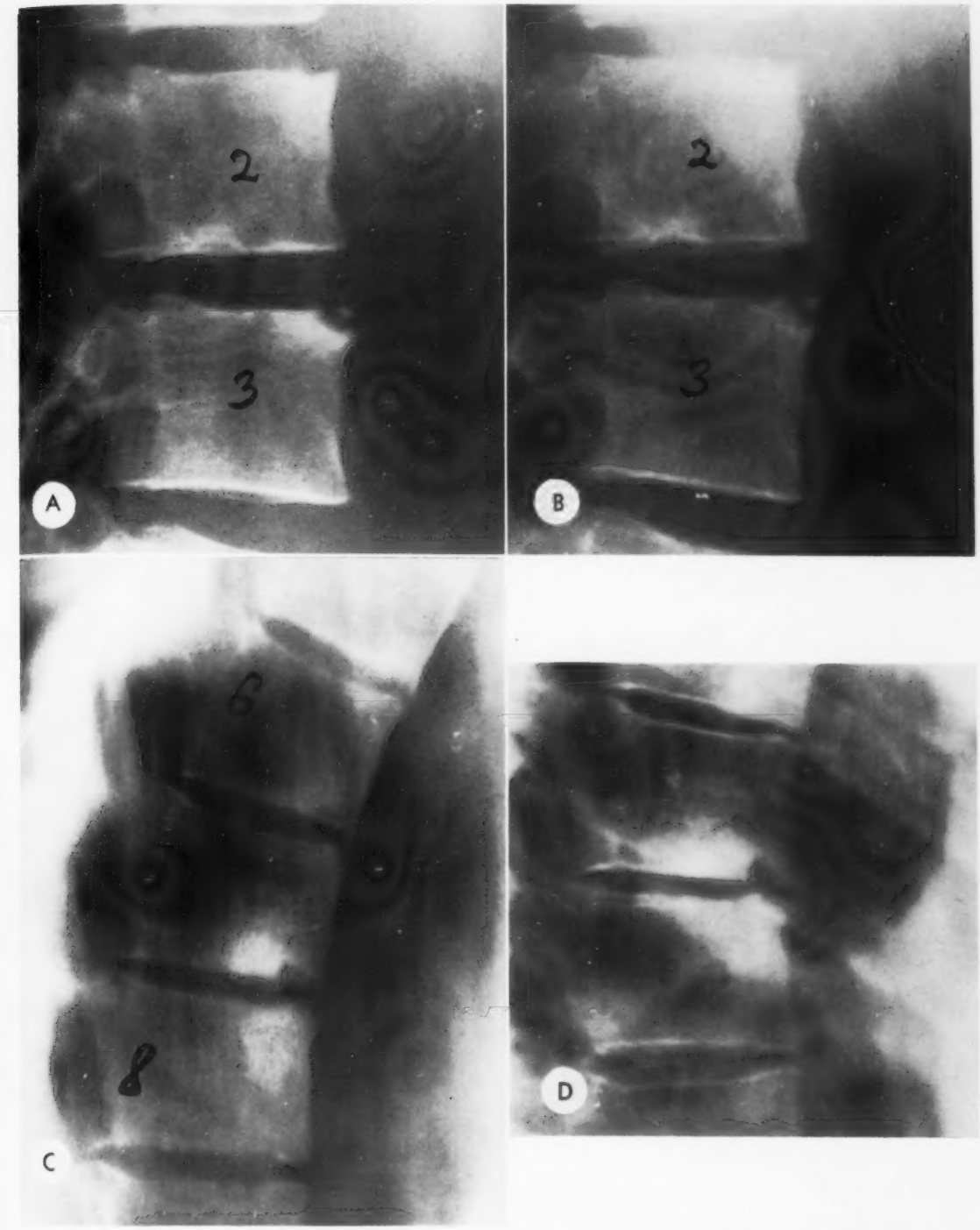

FIG. 1. D. L. (U.#147-64-36), a forty-eight year old female with rheumatoid spondylitis and a four year history of lower dorsal and upper lumbar pain. No steroid therapy. (*A*) Lateral view of the lumbar spine showing erosion of the upper anterior margin of L_3 with adjacent sclerosis. Similar but less marked changes are present in the upper anterior margin of L_2. (*B*) Four years later there has been only slight progression. (*C*) Lateral view of the dorsal spine showing an irregular defect with adjacent sclerosis in the lower anterior margin of T_7. Similar but less marked defects are present in T_5 and T6. (*D*) A detail view of T_7 four years later demonstrating a very slow rate of progression.

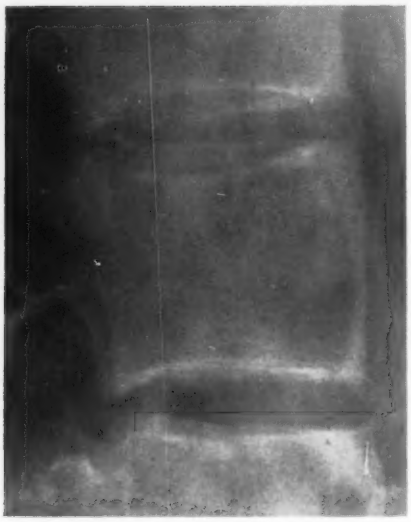

Fig. 2. M. K. (U.#30-94-57), a forty year old male with a seventeen year history of rheumatoid spondylitis. This lateral view of the lumbar spine reveals sclerosis of the anterior corners of L2 and the adjacent corners of L1 and L3. In addition, erosion of the upper anterior corners of L2 and L3 has resulted in the formation of pointed spurs. No steroid therapy.

with this type of process and, in addition, we found 2 cases in patients with peripheral rheumatoid arthritis (Table 1). There were several others who had definite but minimal sclerosis and erosion; they were not included.

Although we were unable to substantiate the statement of Romanus and Ydén that these lesions occur in the majority of cases, we were impressed by the long period of time that intervened between roentgenographic studies in many patients; so they may be correct in their assumption that this stage of the disease, which may be transient, is overlooked.

ROENTGENOGRAPHIC APPEARANCE

The roentgenographic lesions are of two main types which initially seem quite similar but differ in that one progresses to involve the entire extent of the cortical plates of the adjacent vertebral bodies while the second, conceivably an abortive manifestation, seems to remain localized to the anterior corner of a vertebral body (Fig. 1, *A–D*). They both begin as a tiny focus of destruction near the upper or lower anterior corner of the vertebral body associated with varying degrees of sclerosis of the adjacent bone. This may result in sharpening of the corner and a loss of its normal forward convexity so that it projects as a pointed spur (Fig. 2). This process, according to Romanus and Ydén, is responsible for the square vertebra characteristic of rheumatoid spondylitis. This type of destruction was infrequent in our material. Some patients exhibit a small area of destruction without any associated bony sclerosis (Fig. 6). In others the erosion is minimal and the lesion is characterized chiefly by sclerosis (Fig. 7, *A* and *B*).

In other instances, the initial lytic area spreads and the entire surface of the adjacent cortical plates is destroyed. After this occurs, the process seems to remain stationary and little or no further progress is recognizable roentgenographically (Fig. 3, *A-C*).

Two patients of this group, both females, were classified as having peripheral rheumatoid arthritis rather than rheumatoid spondylitis, and they exhibited some differences in the roentgenographic appearance and course of the disease. In these patients, a small initial lytic focus was not observed and the first demonstrable lesion was extensive, involving all of the cortical plates of both adjacent vertebral bodies with rapid complete destruction of the intervertebral disk (Fig. 4, *A–C;* and 5, *A–D*). The lesion then progressed to more or less complete healing so that the end stage resembled the healed stage of tuberculous spondylitis with no remaining trace of the intervertebral disk or adjacent body cortical plate.

ATLANTOAXIAL SUBLUXATION

This unusual complication occurred in a patient with peripheral rheumatoid arthritis who also had a destructive process involving the interspace between the fifth and sixth cervical vertebrae (Fig. 5, *C* and *D*). Laminagraphic studies also revealed evidence of bone erosion of the surface of the odontoid and the anterior arch of the

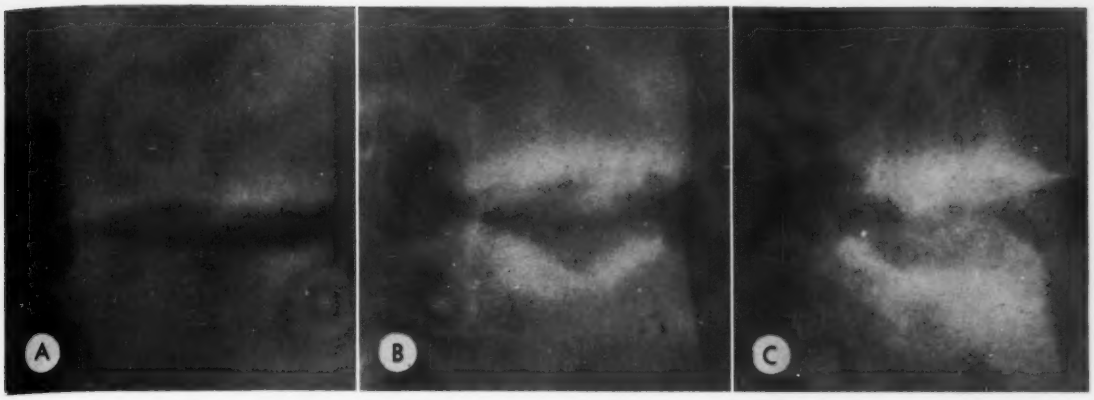

Fig. 3. H. W. (U.#06-67-39), a forty-one year old female with a sixteen year history (in 1960) of rheumatoid spondylitis. (*A*) 1951: A lateral view of L1 and L2 after two years of steroid therapy revealing minimal erosion and sclerosis of the anterior portions of the cortical plates of the adjacent vertebral bodies. (*B*) Eight months later the process has spread to involve the entire intervertebral disk and adjacent vertebral bodies. (*C*) Almost three years after *B* there is only minimal progression. The clinical condition was essentially the same and she has continued to receive cortisone therapy.

first cervical vertebra. Although she complained of severe neck pain, there was no clinical evidence of cord compression. She was treated with a neck brace and has been doing well now for the four years since the discovery of the subluxation. A second patient, not included in this series, with peripheral rheumatoid arthritis and less severe atlantoaxial subluxation, also complained of neck pain and had no neurologic findings. A third patient with rheumatoid spondylitis, also not included, showed evidence of C1–C2 subluxation but no destruction was seen.

Although there are numerous reports describing atlantoaxial dislocation due to acute cervical or nasopharyngeal inflammations,[6] there are relatively few recorded

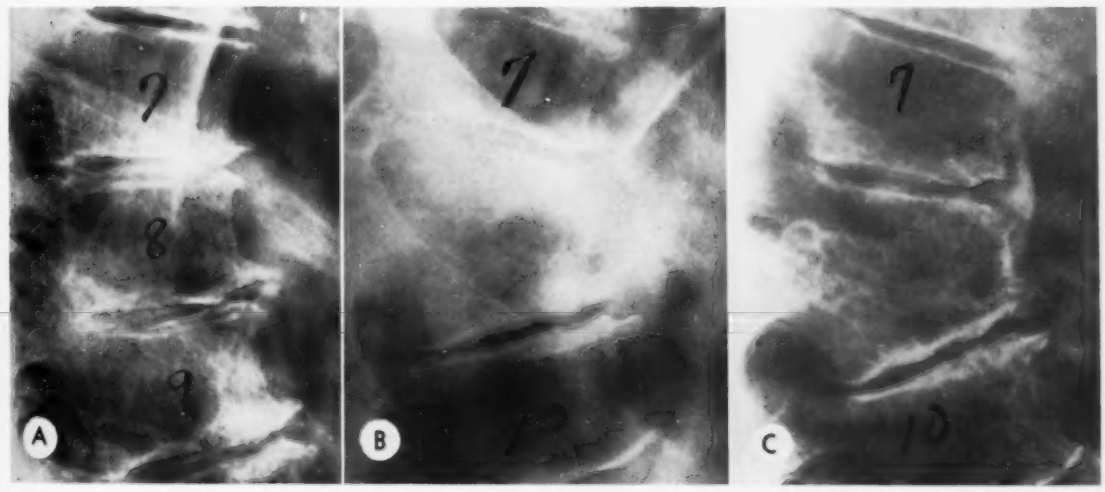

Fig. 4. I. P. (U.#08-89-67), a fifty-eight year old female with a twelve year history (in 1960) of peripheral rheumatoid arthritis. She was treated with ACTH for one month in 1952 and with meticorten from February 7, 1957 to February 23, 1957. (*A*) A lateral view of the thoracic spine, taken in February, 1957 because of severe lower dorsal pain, was interpreted as showing osteoarthritis. (*B*) Seven weeks later there is complete destruction of the intervertebral disk between T8 and T9. Tuberculin test was negative (1:1,000); streptococcal agglutination test was positive. No antibiotic therapy was given. (*C*) A roentgen study three years later shows a healed state with fusion of T8 and T9 and complete disappearance of the intervertebral disk.

Fig. 5. N. B. (U.#03-75-64), a forty-eight year old female with an eleven year history (in 1956) of peripheral arthritis. She had been treated intermittently with cortisone for seven years. (*A*) A lateral view of C6 and C7 taken in 1954 and interpreted as showing a narrow disk space. (*B*) Lateral roentgenogram of C6 and C7 taken three years later because of severe neck pain shows almost complete destruction of the intervertebral disk and the cortical plates of adjacent vertebral bodies. (*C*) Roentgenogram showing the normal appearance of the atlantoaxial joint in 1954. (*D*) In 1957 there was marked posterior and upward displacement of the odontoid and the body of C2.

in patients with rheumatoid spondylitis or rheumatoid arthritis. Purser and Sharp[17] stated that they had seen 17 examples of atlantoaxial dislocation occurring in patients with ankylosing spondylitis and 23 instances in patients with rheumatoid arthritis. They suggested that many cases may be overlooked by failing to take lateral roentgenograms of the cervical spine in flexion. According to Watson-Jones,[23] the hyperemia accompanying inflammations results in softening of the atlas. This may be sufficient to allow detachment of the transverse ligament, which is mainly responsible for maintaining normal relationships between the first and second cervical vertebrae. Margulies *et al.*[13] described atlantoaxial dislocation producing a Brown-Séquard syndrome in a patient with rheumatoid arthritis. Microscopic examination of bone removed at the time of surgery revealed replacement of marrow spaces with fibro-collagenous tissue containing a dense inflammatory infiltrate. Rand[19] also reported a patient who had ankylosing spondylitis with a Brown-Séquard syndrome due to atlantoaxial dislocation, but the association of the two conditions was not recognized. Although the onset of neurologic symptoms may be abrupt and even sudden death may occur, in 85 to 90 per cent the only symptom is painful torticollis.[23] Pratt[16] in 1959 reported 3 more examples and was able to collect 53 cases from the literature (including the 40 mentioned by Purser and Sharp). More recently, Martel[14] described 29 additional cases and he also emphasized that they are usually overlooked unless lateral views of the cervical spine in flexion are obtained.

DISCUSSION

The 11 cases of Marie-Strümpell arthritis with destructive lesions of the spine have certain features in common. The destruction begins anteriorly and remains relatively stationary or it may progress to involve the entire articular surface of the vertebra. The disk space is not always narrowed. In all of these cases there was evidence of disease of the sacroiliac joints, and the patients had had symptoms for one year or more. Four of 11 patients were females, which is a higher incidence than usually is encountered in rheumatoid spondylitis, but the series is too small to be significant. Most of the patients complained of moderate to severe pain in the region of the destructive process, but a few patients had no symptoms referable to the site of the lesion.

The roentgenographic appearance may be indistinguishable from that produced by tuberculosis, Staphylococcus, typhoid, parathyphoid or Brucella infection of the vertebra. There are a few minor differences in that, with bacterial infections, narrowing of the intervertebral disk usually precedes roentgenographic evidence of bone destruction, while in rheumatoid disease bone erosion is frequently demonstrable without disk narrowing and occasionally even with disk widening (Fig. 3, *A–C*). In some cases disk narrowing develops later (Fig. 7, *A* and *B;* 8, *A* and *B*). Paravertebral masses are frequently associated with bacterial osteomyelitis of the spine, while they are rare in rheumatoid disease. The rate of destruction of the intervertebral disk classically is rapid in pyogenic osteomyelitis and relatively slow in tuberculous infection,[15] but in both it progresses more rapidly than in the majority of our patients. Wholey *et al.*[25] also stated that the lesions in their 10 patients failed to show the progression that would be expected of other infectious or granulomatous processes. Once the entire intervertebral space and cortical plates of adjacent vertebral bodies were involved, the process seemed to remain static, except for the 2 patients in whom spontaneous healing occurred. Tuberculous skin testing was done in only 1 patient in this series (Case IV, Table 1; Fig. 4) and that was negative at 1:1,000 dilution. No patient received specific antituberculous therapy.

Fig. 6. F. D. (U.#79-94-59), a thirty-six year old male with an eighteen year history of rheumatoid spondylitis. On this roentgenogram made in 1954 note a destructive lesion involving the intervertebral disk between T5 and T6 and destruction of the adjacent bone anteriorly. The patient had no back symptoms at the time of this study. Cortisone therapy had been given two and four years earlier, for ten days each time, because of uveitis.

The role of steroid therapy in the pathogenesis is difficult to assess. That it is not the sole causative factor is evidenced by the 5 patients in this series who were not treated with this type of drug. There are also reports of several cases in the literature which were not treated with steroids.[19] Of the 8 patients who did receive steroid therapy, 3 had received only one short course (two to three weeks) of cortisone or ACTH two or three years prior to the discovery of the osteolytic spinal lesion. On the other

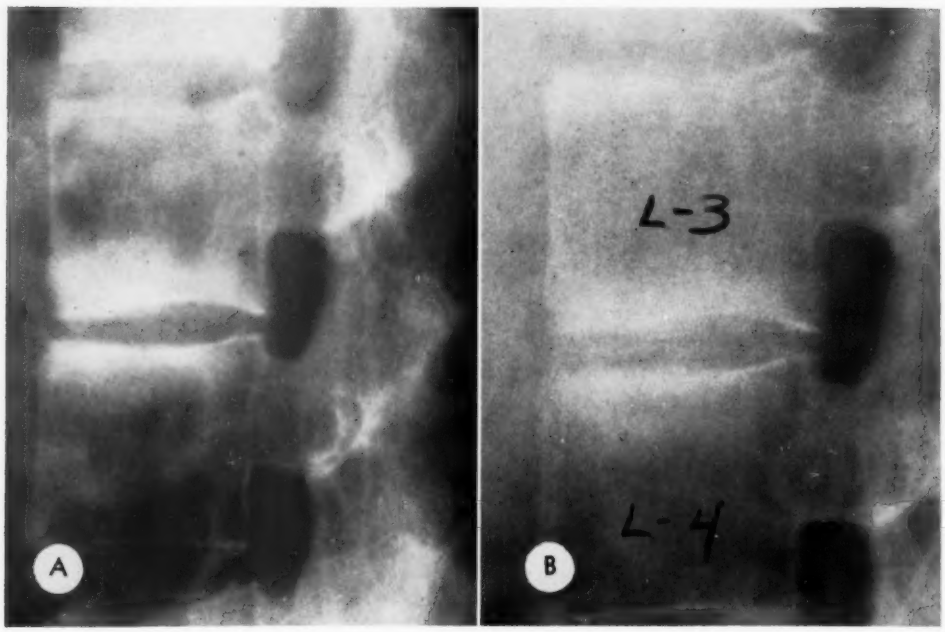

Fig. 7. H. D. (U.#01-85-79), a twenty-nine year old male with a nine year history of rheumatoid spondylitis. A ten week course of cortisone had been given in 1950. (*A*) A lateral view of the lumbar spine in 1952 showing sclerosis of the L_3 and L_4 vertebral bodies adjacent to the intervertebral disk. Note the minimal erosion of the anteroinferior corner of L_3 and the anterosuperior border of L_4. (*B*) Two years later the vertebral margins are smoother and the sclerosis has diminished. This process may be the pathogenesis of the square vertebra seen in rheumatoid spondylitis.

hand, 3 patients were on cortisone continuously for two years, eight years and nine years, respectively. One possible etiologic mechanism is the potentiation of any type of infection, particularly tuberculosis, because of suppression of the body's immune mechanism by the steroid. Steiner[22] has reported experiences with patients on prolonged cortisone therapy in whom at autopsy multiple infective lesions of the spine were demonstrated. During life, these patients were asymptomatic and roentgenographically negative. Most of our cases which had extensive destruction were treated with steroids[3] with the exception of Cases 1 and 11. On the other hand, 2 patients were continued on cortisone after the spinal lesion was demonstrable without any clinical or roentgenographic progression of the disease.

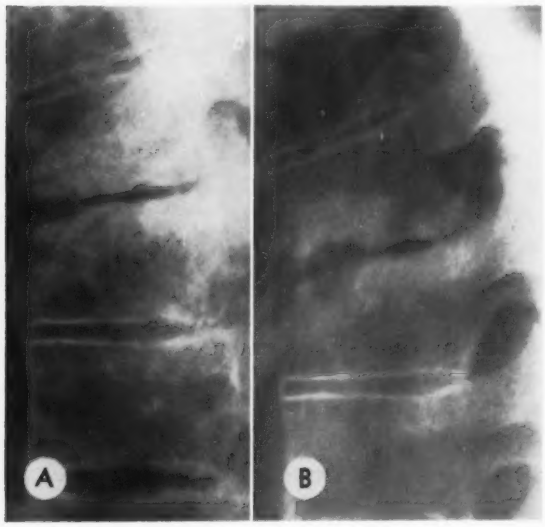

Fig. 8. H. E. (U.#11-94-82), a fifty-three year old male with a history of rheumatoid spondylitis for twelve years. He was treated with cortisone for two years. (*A*) A lateral view of the thoracic spine demonstrating a destructive lesion involving the 7th and 8th dorsal vertebral bodies adjacent to the intervertebral disk. (*B*) One year later the destructive process had extended to involve the entire extent of the intervertebral disk and the adjacent cortical plates.

The destruction and disappearance of the femoral head that may occur during steroid therapy[2,11,18] suggest that a similar phenomenon could occur in the spine. Murray[15] described a patient with purpura who was treated with 40 mg. of prednisolone daily for six days and who developed a similar destructive lesion of the anterior portion of the intervertebral disk and adjacent bone. Although the presumptive diagnosis was tuberculosis and the patient promptly healed on antituberculous therapy, bacteriologic verification was never obtained. Ansell[2] reported a patient with acute pancreatitis and hepatitis who developed a destructive vertebral lesion after thirteen days of treatment with ACTH. This was also assumed to be tuberculosis and was treated accordingly, although the etiologic agent was never discovered. It is possible that most of these lesions may represent rheumatoid involvement of the spine with granulation tissue similar to that in the autopsied patient reported by Baggenstoss *et al.*[3]

CONCLUSION

1. Thirteen patients, 11 with rheumatoid spondylitis and 2 with peripheral rheumatoid arthritis, with destructive lesions of the vertebral bodies are reported.

2. The roentgenographic appearance of the lesion simulates bacterial osteomyelitis of the vertebra but the clinical and roentgenographic course fails to show the progression usually seen in bacterial infection.

3. The relationship to tuberculosis, pyogenic infections and steroid therapy and rheumatoid disease is discussed.

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OSTEOLYSIS OF THE ACROMIAL END OF THE CLAVICLES IN RHEUMATOID ARTHRITIS*

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DESTRUCTIVE changes in the outer end of the clavicles have been observed in 11 patients with advanced rheumatoid arthritis. Since these osseous manifestations have not been adequately described previously, it is the purpose of this paper to present these findings.

REVIEW OF THE LITERATURE

A survey of the medical literature of the past seventy-five years brought to light very little information on the clavicular changes in rheumatoid arthritis. In over 150 books and articles dealing with rheumatoid arthritis and disorders of the shoulder, we found only 2 reports[15,22] which mentioned significant changes in the outer ends of the clavicles.

In 1949 Layani *et al.*[22] published the case of a sixty-five year old woman who had had rheumatoid arthritis involving many peripheral articulations for a seventeen year period. Joint motion was so painful that she was confined to bed or a chair. On physical examination the elbows were fixed, and there was shortening of the phalanges. Roentgen examination showed severe demineralization of the entire skeleton, with advanced changes due to rheumatoid arthritis in the hands, wrists, knees and hips. Tapering of the outer ends of the clavicles and widening of the acromioclavicular joint spaces were noted. In 1953 Jacobs *et al.*[15] described the case of a sixty-three year old woman who had suffered from widespread rheumatoid arthritis for twenty-eight years. Roentgen examination showed striking arthritic changes in the hands, wrist, elbows, knees, ankles and feet. Jacobs and his co-workers stated that there was a loss of bone substance of the outer end of the left clavicle. The illustrations in this article show that this patient also had a similar involvement of the outer end of the right clavicle.

Kienböck's comprehensive "Röntgendiagnostik der Knochen und Gelenkkrankheiten" does contain a description of 3 patients (No. 120, 122 and 123) who had destructive changes in the outer ends of the clavicles, which were, in all likelihood, secondary to rheumatoid arthritis. Unfortunately, Kienböck diagnosed the first case as rheumatoid tuberculosis, the second as rheumatoid tuberculosis (Poncet) and the third as rheumatoid tuberculosis with caries sicca. It should be mentioned that in Kienböck's era the Austrian pathologists seriously considered tuberculosis as the etiology of the affection we now call rheumatoid arthritis.[1,3,31]

That acromioclavicular joint changes may take place in rheumatoid arthritis was known by Oppenheimer,[30] who in 1944 stated that roentgenograms may demonstrate narrowing of the joint space and erosion of the articular surfaces. He found that the arthritic process may progress to bony ankylosis, but in other patients the destructive process stops, and sclerotic areas appear in the articular areas, thereby simulating osteoarthritis. In 1954 Laine *et al.*[21] studied the records of 277 hospitalized patients with rheumatoid arthritis and found that 57.4 per cent had shoulder symptoms. Only one of these patients, however, had acromioclavicular arthritis, and the roentgen findings were not described. In Schinz's "Roentgen-Diagnostics" "erosion of the articular surfaces" is mentioned in the text. The authors of two books dealing exclusively with arthritis of the shoulder[24,41] do not discuss the alterations of the clavicle. Three other recent

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TABLE I

PERTINENT FINDINGS IN 11 PATIENTS WITH CLAVICULAR OSTEOLYSIS DUE TO RHEUMATOID ARTHRITIS

Case No.	Age*	Sex	Duration Rheumatoid Arthritis (yr.)	Shoulder Symptoms and Signs	Other Joint Symptoms	Physical Findings in Other Joints	Roentgen Findings in Other Joints	Laboratory Data
I	47	M	18	None	Progressive deformity and disability of peripheral joints	Marked flexion deformities and contractions of all peripheral joints. Fingers in ulnar deviation. Wrists fused. Decreased mobility of temporomandibular joints	Knees, elbows, hands and wrists, feet and ankles: demineralization flexion contractures with ulnar deviation, destruction of articular cortices, subluxation of metacarpophalangeal and knee joints. Otto pelvis, fusion of sacroiliac and manubriosternal joints. Marked deformity of femoral heads	E.S.R. 80 Latex fixation test positive
II	65	F	45	None	Moderate pain and progressive deformity in hands, feet, ankles, knees, wrists, elbows and hips	All distal joints deformed, with contractures at elbows. Right wrist fused. Severe ulnar deviation of fingers. Hip flexion only to 20°	None available	E.S.R. 32. Latext fixation test positive
III	67	M	6	Limitation of motion and deformity of shoulders	Progressive deformity of small joints, and limitation of motion of all large joints, including knees and elbows	Decreased motion in eblows, knees and ankles. Knees swollen and warm. Ulnar deviation of fingers. Subcutaneous nodules over elbows and wrists. Spine moderately stiff. General muscle atrophy	Hands and knees: demineralization, gross erosion and destruction of articular surfaces, numerous cyst-like rarefactions, narrowed joint spaces. Subluxation of metacarpophalangeal joints	E.S.R. 38
IV	31	F	9	Progressive ankylosis of shoulders	Progressive stiffness and flexion contractures of knees, ankles, elbows, hips, and jaws	Extremities emaciated, with ulnar deviation of fingers. Flexion contractures and markedly restricted motion of joints	Right wrist and knee fused. Severe periarticular demineralization, marked erosion and destruction of all articular surfaces; cyst-like areas of rarefaction, and narrowed joint spaces in knees, ankles, elbows and hips	None available
V	47	F	24	None	Progressive deformity and disability of all peripheral joints	Marked flexion deformities and contractures of hands, ankles, feet and knees, with limitation of motion mainly in hands. Ulnar deviation of fingers. Fusion of wrists	Ankylosis of carpal bones. Advanced demineralization, articular destruction, and subluxation of phalanges of hands and feet; joint space narrowing in hands, feet and knees. Pseudocyst in head of fifth metatarsal	E.S.R. 41
VI	58	F	26	Limited motion of shoulders	Progressive flexion contractures of left hip, wrists and knees	Contractures, limitation of motion and crepitation in left hip, knees and wrists. Subcutaneous nodules over elbows	None available	None available
VII	45	F	23	Frozen right shoulder	Progressive painful stiffness and deformities of many joints, most marked in hands	Joint deformities in hand with ulnar deviation of motion, and small subcutaneous nodules. Elbows stiff. Fluid in knee joints	Elbows, wrists, hands, knees, ankles, feet: flexion contractures with ulnar deviation, demineralization, destruction of articular cortices, subluxation of metacarpophalangeal joints	E.S.R. 42. Latext fixation test positive. Sheep cell agglutination positive

Case No.	Age*	Sex	Duration of Rheumatoid Arthritis (yr.)	Shoulder Symptoms and Signs	Other Joint Symptoms	Physical Findings in Other Joints	Roentgen Findings in Other Joints	Laboratory Data
VIII	61	M	18	None	Progressive deformity and disability of hands, wrists, feet and ankles	Marked flexion contractures and deformities of hands and elbows. Subcutaneous nodules	Knees: demineralization, narrow joint spaces, destruction of articular cortices, subluxation	E.S.R. 41. Latex fixation test positive
IX	49	M	14	Limitation of motion of left shoulder and atrophy of left shoulder girdle	Chronic progressive deformity and disability of right fingers, ankles, and knees	Wrists, knees, ankles, first and second right fingers: ulnar deviation, fusiform swelling, flexion contractures	Hands, wrists, knees, ankles, and feet: loss of articular cortices, joint space narrowing, demineralization, cyst-like areas of rarefaction, periarticular swelling	E.S.R. 150. Sheep cell agglutination positive
X	35	F	22	None	Progressive deformity and disability of peripheral joints	Flexion contractures of elbows, wrists and fingers. Fusiform deformity and ulnar deviation of fingers	Knees, feet, elbows: flexion contractures, narrow joint spaces, demineralization pseudocyst formation, destruction of articular cortices	E.S.R. 22
XI	56	M	10	None	Progressive deformity and disability of hands, wrists, elbows, knees and feet	Hands, wrists, elbows, knees, feet: flexion contractures, periarticular Ulnar deviation of fingers	Hands and wrists: narrow joint spaces, demineralization, loss of articular cortices, cyst-like areas of rarefaction	E.S.R. 116. Latex fixation test positive

* Age of patient at the time of last visit.

publications[14,43,55] contain roentgenograms demonstrating the clavicular tapering, but without comments from their authors. Erosions of the acromial ends of the clavicles have been described in 2 patients with severe psoriatic arthritis.[17,42] Unilateral acromioclavicular arthritis has also been observed in one patient with long-standing Marie-Strümpell arthritis.[5] Involvement of the sternoclavicular joints by rheumatoid arthritis has been studied by several authors,[18,35,44,48] and this affection will not be discussed in the present paper.

CLINICAL MATERIAL

In our study of 11 patients with clavicular changes, the average age was fifty-one years, with a range of thirty-one to sixty-seven. There were 6 women and 5 men. The average duration of the arthritic symptoms was 19.5 years, with a minimum of six and a maximum of forty-five years (Table 1). Five of the 11 patients had complaints referable to the shoulder, but the medical records did not show implication of the acromioclavicular joints. The usual symptoms were pain and stiffness in multiple peripheral articulations, and the common physical findings included ulnar deviation of the fingers, flexion deformities, decreased motion and swelling of the joints. In 9 patients the erythrocyte sedimentation test had been done, and in each the rate was elevated, with a variation from 22 to 150. In 6 patients a laboratory test for rheumatoid arthritis[32,38,50] had been carried out (sheep cell agglutination or latex fixation), and in 5 it was positive. The patient with the negative latex fixation test (Case x) had characteristic clinical and roentgen manifestations of long-standing peripheral rheumatoid arthritis, the disease having started at thirteen years of age. This test had been done only once, when she was thirty-five years old, and at that time she was clinically in a state of remission. Her sedimentation rate (Westergren) was only 22 mm. per hour, and this lack of activity probably explains the low titer.[38]

TABLE II

ROENTGEN FINDINGS IN OUTER END OF CLAVICLE IN 11 PATIENTS WITH CLAVICULAR OSTEOLYSIS DUE TO RHEUMATOID ARTHRITIS

Case No.	Right Clavicle	Left Clavicle	Acromioclavicular Angle	
			Right	Left
I	Marked loss of bone substance inferiorly, with widening of the joint space (15 mm.). Demineralization	Demineralization, with narrowing of the joint space and an osteophytic spur	28°	42°
II	Slight demineralization. Irregular margins. Decreased joint space	Loss of bone substance inferiorly, with small area of sclerosis	30°	10°
III	Truncated with irregular margins	Loss of bone substance inferiorly, with widening of joint space	25°	31°
IV	Distal tapering with loss of bone substance inferiorly		44°	27°
V	Loss of bone substance inferiorly, producing tapering with widening of the joint space (15 mm.). Progressive changes over 12 year course		33°	45°
VI	Loss of bone substance inferiorly, producing tapering, and widened joint space. Progressive changes over 9 year course		37°	66°
VII	Loss of bone substance producing tapering		52°	29°
VIII	Bulbous appearance with scalloping, progressing to "ace-of-spades" appearance over 2 year course	Progressive scalloping	40°	32°
IX	No changes	Loss of bone substance inferiorly	0°	45°
X	Notching	No changes	32°	14°
XI	Scalloping	Loss of bone substance inferiorly producing tapering, with demineralization	35°	28°

ROENTGEN FINDINGS

The roentgenograms in 7 of our patients revealed a striking loss of bone substance at the acromial end of each clavicle. The other 4 cases showed only cortical erosion and pseudocyst formation (Table II). In severe cases the outer end of the clavicle often had a tapered appearance, but bone destruction was more marked inferiorly than superiorly. Accompanying changes of the acromion process, characterized by demineralization and articular erosion, were less marked and more difficult to recognize. The acromioclavicular joint space was definitely widened in 6 patients. Two patients had unilateral narrowing of this joint space due to secondary degenerative alterations. The osseous structures of the shoulder girdle showed severe osteoporosis. Involvement of the glenohumeral joint by rheumatoid arthritis was seen in 9

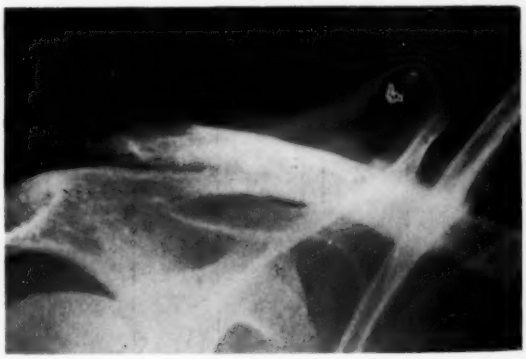

FIG. 1. Case V. (1947) Roentgenogram of a thirty-five year old woman who has had rheumatoid arthritis for twelve years showing a normal outer end of the right clavicle.

FIG. 2. Case v. (1955) Roentgenogram showing loss of bone substance of the outer end of the right clavicle, especially inferiorly.

FIG. 4. Case vi. Roentgenogram of a fifty-eight year old woman who has had rheumatoid arthritis for twenty-six years showing considerable tapering of the outer end of the right clavicle.

of the 11 patients, and many of the peripheral joints showed evidence of long-standing rheumatoid arthritis.

In one patient (Case v, Fig. 1, 2 and 3) we were able to follow the unfavorable progress of the clavicular lesion from a normal status in 1947 to a definite loss of bone substance of the acromial end in 1953. By 1955 the tapering had increased, and in 1959 there was a 15 mm. gap at the acromioclavicular joints due to continued clavicular bone loss. Another patient (Case vi, Fig. 4) had progressive tapering of the outer ends of the clavicles over a nine year interval; and still another (Case vii, Fig. 5) showed a beaked appearance of the outer end of the right clavicle with a fifteen year history of rheumatoid arthritis.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of a destructive lesion of the outer end of the clavicle, there are many conditions to consider: normal variation of the acromial end of the clavicle, cleidocranial dysostosis, osteogenesis imperfecta, hyperparathyroidism, acromioclavicular dislocation, post-traumatic osteolysis of the clavicle, surgical resection of the outer end of the clavicle, inflammatory diseases, degenerative changes, scleroderma, acroosteolysis and neoplastic lesions.

Normal Clavicle. The normal clavicle was examined in over 300 adult patients to evaluate the roentgen appearance of the acromial end. This study confirmed the fact that there is considerable variation in the configuration of the outer end of this bone. Although many individuals had expansion of this segment of the clavicle, we found only one patient who had any appreciable tapering of this region. On the anteroposterior roentgenogram of the shoulder, the plane of the normal acromio-

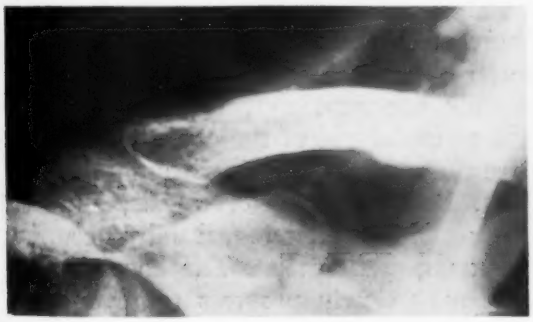

FIG. 3. Case v. (1959) Roentgenogram showing further tapering of the outer end of the right clavicle and marked widening of the acromioclavicular joint space.

FIG. 5. Case vii. Roentgenogram of a thirty-seven year old woman who has had rheumatoid arthritis for fifteen years showing a beaked appearance of the outer end of the right clavicle due to bone loss both superiorly and inferiorly.

clavicular joint is usually angulated with respect to the sagittal plane, so that the inferior aspect of the articulation is more medially situated than the superior part. It is especially this type which must be differentiated from erosion of the inferior aspect due to rheumatoid arthritis.

DePalma[4] used the magnitude of this angle as a criterion to classify all clavicles into 3 categories. Type 1 had an average angle of 16°, Type 2—24.6° and Type 3—36.1°. Of the 66 clavicles studied by De Palma 89 per cent were either Type 1 or Type 2, and only 11 per cent were Type 3. In our 11 patients with rheumatoid arthritis, there were only six clavicles that fell into Types 1 and 2, while 8 were of Type 3 variety. These figures indicate a reversal of the normal distribution. Seven clavicles had such high angles (ranging from 40° to 66°) that they could not be placed in any of DePalma's categories. One clavicle had an angle of 0°, indicating that it was parallel to the sagittal plane. The average angle of the 22 clavicles in our study was 33° (Table II).

The normal acromioclavicular joint space is 2–5 mm. in diameter,[30] but in rheumatoid arthritic involvement this gap may be increased to 15 mm. (Case 1).

Osteogenesis Imperfecta. In osteogenesis imperfecta one may see a slender clavicle bilaterally, with tapering of the outer ends. In contrast to stiff joints, however, these patients often have hypermobile articulations. The presence of fresh fractures, deformities due to old fractures, and bowing of the long bones will aid in the differential diagnosis. Blue sclerae are usually present, and there is sometimes a family history of this disorder.

Hyperparathyroidism. Erosive changes in the outer ends of the clavicles are important findings in the roentgen diagnosis of hyperparathyroidism.[9,28,47] In both hyperparathyroidism and rheumatoid arthritis, the loss of bone substance is characteristically bilateral, and there is demineralization of the regional osseous structures. As a rule neither condition produces clavicular pain, but there may be a local tumefaction at the site of the lesion in hyperparathyroidism. In hyperparathyroidism we have not observed sharp tapering of the clavicle. In patients with hyperparathyroid bone disease, there will be subperiosteal cortical resorption of the phalanges,[8,34] and most of them will have definite skull changes,[7] including demineralization of the skull tables and loss of the lamina dura. The presence of renal calculi or nephrocalcinosis will favor the diagnosis of hyperparathyroidism.

Acromioclavicular Dislocation. The usual case of an acute acromioclavicular dislocation[46] will not constitute a difficult problem in differential diagnosis, as there will be a history of trauma, and the patient will complain of pain in the joint region. On both physical and roentgen examination, the outer end of the clavicle will be displaced superiorly, a finding not present in rheumatoid arthritis. We have observed several cases of chronic acromioclavicular dislocations in which there was a considerable gap in the acromioclavicular joint space due to rupture of the acromioclavicular ligaments, and this finding may resemble that seen in rheumatoid arthritis. Factors to be considered, however, in differential diagnosis include the past history of a severe injury, the unilaterality of the lesion, the presence of bone sclerosis or deformity secondary to an old healed fracture. Often there is also evidence of calcification or ossification of the torn coracoacromial and acromioclavicular ligaments.

Post-Traumatic Osteolysis. Post-traumatic osteolysis[16,54] involves the clavicle more often than any other bone, and the outer end is the site of predilection. It is readily apparent that the dissolution of the acromial end of the clavicle will simulate the changes seen in rheumatoid arthritis. Post-traumatic osteolysis, however, is a unilateral affection, usually occurring in young adult males. The bone destruction may slowly progress to involve the adjacent scapula and ribs, while costal extension does not occur in rheumatoid arthritis. A biopsy may be necessary for diagnosis, and the histologic appearance is fre-

quently that of hemangiomatosis.[10] It might also be mentioned in this connection that unilateral erosion of the outer end of the clavicle has been described as an occupational disease in men using compressed air tools,[6] evidentally on the basis of repeated minor injuries.

Surgical Resection. A resection of the outer end of the clavicle[51] may result in a roentgen appearance resembling the clavicular deformity of rheumatoid arthritis. In one of our patients (Case XI) with rheumatoid arthritis, the clavicular defect was erroneously interpreted as being a postoperative sequel. A sharply tapering clavicle is not apt to be of surgical origin.

Tuberculosis. Tuberculosis may produce erosion of the outer end of the clavicle, thereby simulating the lesion seen in rheumatoid arthritis. In some cases of clavicular tuberculosis, moreover, the acromion is not appreciably involved by the destructive process, and there may be no soft tissue swelling and very little discomfort.[39,46] Unilateral involvement would favor the diagnosis of tuberculosis, while polyarthritis would favor rheumatoid arthritis. If the acromion is severly involved, if there is a painful soft tissue mass and if there is a draining sinus, tuberculosis is undoubtedly present. Glenohumeral joint involvement is not apt to be associated with a clavicular lesion in tuberculosis, but this association is common is rheumatoid arthritis. Aspiration or biopsy of the lesion may be required, if tuberculosis is suspected.

Other Inflammatory Lesions. Inflammatory lesions other than tuberculosis rarely affect the clavicle; however, this has been reported in cases of pyogenic osteomyelitis, Brodie's abscess, mycotic infection, gumma and eosinophilic granuloma involving the bone.[2,11,36] All five lesions are unilateral and will often be accompanied by soft tissue swelling, but the latter is absent in rheumatoid arthritis. Subperiosteal new bone formation is present in a pyogenic osteomyelitis and may also be seen in eosinophilic granuloma, but this change will not be evident in rheumatoid arthritis. In the case of gumma, the Wassermann test will be of considerable diagnostic value.

Degenerative Arthritis of the Acromioclavicular Joints. Degenerative arthritis of the acromioclavicular joint is encountered twice as often as all other disorders of this articulation combined.[13,26,27] The roentgen appearance is considerably different from that of rheumatoid arthritis, for there is sclerosis, osteophyte formation and narrowing of the joint space.

Scleroderma. Meszaros mentioned in his recent review on scleroderma that one patient had a loss of bone substance at the acromial end of each clavicle. This osseous abnormality is very rare in scleroderma, and we were unable to find a similar case reported in the literature. Despite the presence of the clavicular lesion, there should not be any difficulty in distinguishing scleroderma from rheumatoid arthritis. The presence of Raynaud's phenomenon, hardened facial skin with immobile expression, ulceration of the finger-tips and dysphagia are clinical features which would favor the diagnosis of scleroderma. The following roentgen features are also useful in supporting this impression: loss of the manual ungual tufts, calcinosis and atony of the esophagus.

Acroosteolysis. There are several reports in the literature indicating that patients with acroosteolysis may have erosions of the outer end of both clavicles.[12,20,52] In these cases the shortening of the fingers may simulate the "opera glass hand" of mutilating rheumatoid arthritis; however, these patients do not have a history of polyarthralgias associated with soft tissue swelling and warmth of the affected articulations. Nor do they have pain or limitation of motion in the larger peripheral joints. The roentgen examination will show a progressive loss of bone substance of the ungual tufts, then of the entire distal phalanges and finally of the middle phalanges, as well. Both hands and both feet are characteristically involved, but the joints are not the first structures to be affected.

Neoplasms. Both primary and secondary neoplasm of the clavicles are rare,[33] and

one would not expect the outer end of both clavicles to be involved in a single patient. The presence of a painful mass will be against the diagnosis of rheumatoid arthritis, and if there is a question of tumor, a biopsy is often indicated.

DISCUSSION

The pathology of the clavicular changes in rheumatoid arthritis is not known, as we do not have any biopsies of this region. However, since at autopsy the outer end of the clavicle is available for examination, we hope that these changes will be clarified in the near future. In all probability the erosion of the clavicle is similar to the loss of bone seen in mutilating arthritis. Here, gross and microscopic examination has demonstrated replacement of the involved bone end, articular cartilage, synovial membrane and a considerable portion of the muscle by fat[23] or fibrous tissue.[53] These pathologic findings evidently represent the residue of a chronic inflammatory process due to rheumatoid arthritis.

SUMMARY

The erosive changes in the outer ends of the clavicles in cases of rheumatoid arthritis have been described. The clavicle is tapered or scalloped due to a loss of bone substance, and both clavicles are often involved. The acromioclavicular joint space may be widened, but the acromion process is not severely affected. This clavicular lesion must be distinguished from bone destruction caused by hyperparathyroidism, tuberculosis, post-traumatic osteolysis and acroosteolysis.

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THE OCCIPITAL VERTEBRA

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ACCORDING to the investigations of Froriep, the basilar process of the occipital bone is formed by fusion of the first three primitive vertebrae (or occipitoblasts), the most caudal of which is the so-called occipital vertebra (Froriep) or proatlas (Albrecht). A theory advanced by Chiarugi in 1895 and later developed by Kollmann in 1905 attributes failure of the distal occipitoblast to fuse with the others as the cause of abnormal bone formations on the external surface of the skull around the occipital foramen. This phenomenon is called the "manifestation of the occipital vertebra."

This malformation, described for the first time at the beginning of the 19th century, has been the subject of numerous studies in normal and comparative anatomy and anthropology. The great interest derives from the fact that the occipitovertebral joint, just as the lumbosacral joint, is in an embryologically unstable region. In the vertebrates, the border between the cranium and the vertebral column moves in the caudal direction and reaches its lowest position in the mammals. In man the instability of this border is observed in cases of assimilation of the atlas and is to be regarded as a progressive variant, while in cases of manifestation of the occipital vertebra it is to be regarded as a recessive trait.

The concept of the manifestation of the occipital vertebra and its limits have not as yet been clearly defined. Schmidt and Fischer[16,17] remark that there is hardly a variant of the occipital bone or of the cranial part of the atlas which has not been attributed to it. We have included: (a) the third condyle; (b) the paracondyloid process; (c) the basilar processes; (d) the accessory bone elements, separate or fused to the foramen magnum; and (e) the transverse fissures of the basioccipital bone.

From the anatomic point of view, this classification is not complete; some variants have been omitted either because they are not demonstrable roentgenologically (such as the division of the foramen of the hypoglossal nerve) or because they can be classified among the anatomic variants (such as the ponticulus posterius which, closing the sulcus of the vertebral artery of the atlas, forms the arched foramen).

We have found only a few articles concerning this malformation in the roentgenologic literature. The first one, published by Hadley[7] in 1948, has been followed by a few others, which deal mostly with isolated observations. Exceptions to this are the publication of McRae and Barnum[14] on 5 paracondyloid processes observed among 25 cases of assimilation of the atlas, the study of Schmidt,[16] carried out partly in the living subject and partly on anatomic material, the report of Fischer[5] and the recent work by Schmidt and Fischer[17] on occipital dysplasia.

The present paper concerns the manifestation of the occipital vertebra based on roentgen examinations of 4,000 consecutive patients. The collected cases of this malformation were 19 in number—a high incidence if we consider the limitations of the roentgen examination, however accurately it is performed.

ROENTGEN TECHNIQUE

The study of the atlanto-occipital region requires precise lateral roentgenograms, without rotation of the head. Any movement modifies the relationships and obscures visualization of the region. The study is completed by using other projections and laminagraphy, which is indispensable and which, in selected cases,

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includes the axial transverse plane. This latter method of examination has proved to be of considerable value for the study of the occipital foramen, since it shows its exact shape and actual dimensions free from the frequently observed distortions and superimpositions brought about by projection factors (Fig. 1, *a* and *b*).

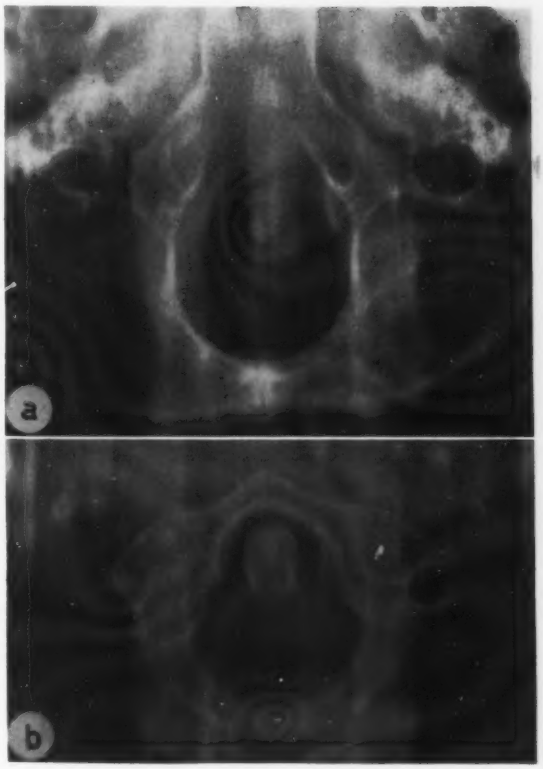

Fig. 1. Axial transverse laminagrams. Sections through (*a*) foramen magnum and (*b*) the atlas.

ROENTGEN FEATURES

THIRD CONDYLE

Early in the last century, Meckel described the third condyle as a median bone formation of exocranial development which detaches itself from the anterior contour of the occipital foramen. The condyle is usually narrow at its base, becoming broader at the level of its inferior surface which bears an articular surface for the apex of the odontoid of the axis or, more rarely, the anterior arch of the atlas. It varies in size; when it shows a pronounced development it can also be observed on plain lateral roentgenograms of the skull (Fig. 2*a*). Hadley[7] mentions specimens 13 and 14 mm. in length and, in the case published by Kamieth,[9] the formation was even bigger. However, as a rule, it is rather small and must be searched for with the aid of laminagraphy (Fig. 2*a'*; and 3, *a-d*). Continuity of the condyle with the basiocipital bone is sometimes indicated by an

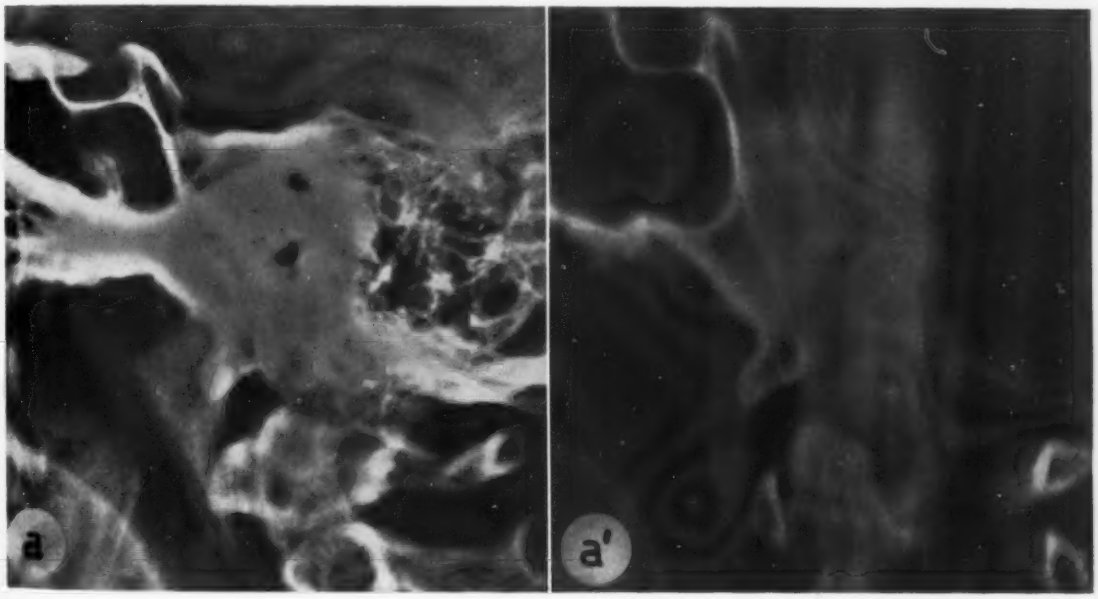

Fig. 2. The third condyle seen on (*a*) a roentgenogram and (*a'*) a laminagram.

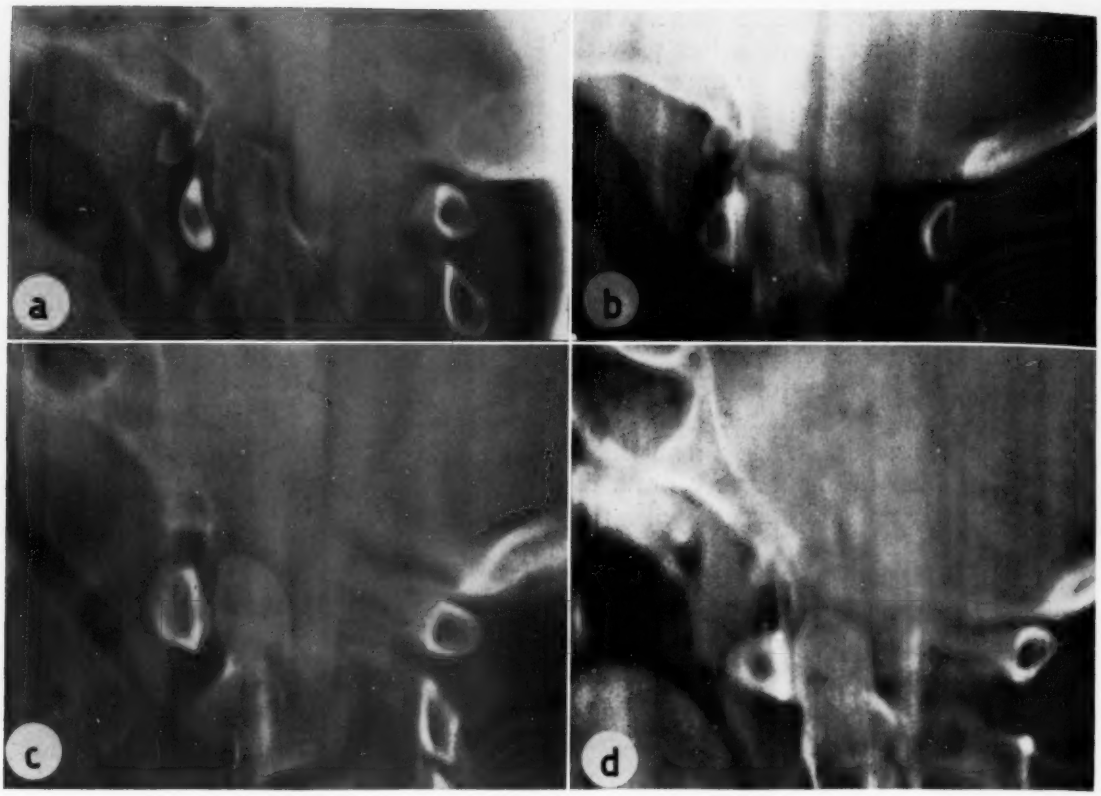

FIG. 3. (*a–d*) Different aspects of the third condyle.

incisura and by a transverse streak of denser bone (Fig. 3, *a* and *b*). The 6 cases we have collected make this the most frequent malformation in our series of manifestations of the occipital vertebra. In 5 cases the anomaly was present in its pure form; in the sixth it was associated with an anomaly of the axis (separate odontoid process). None of our patients had any complaints attributable to the condyle, but it is known that when it is large it may restrict head movements.

According to Kollmann, whose interpretation we will further refer to below, the third condyle is the result of incomplete regression of the hypochordal arch of the occipital vertebra or pro-atlas. Brocher[1] believes that it corresponds to the body of the occipital vertebra as does the odontoid process to the body of the atlas.

PARACONDYLOID PROCESS

In the skull of the adult man, on each side of the occipital condyles, we usually find ridges and rough spots in the bone where muscles or tendons are inserted. When these formations, which embryologically derive from the inferior articular apophysis of the occipital vertebra, are larger than normal, they are termed the "paracondyloid process" or "manifestation of the transverse process of the atlas."[15]

In the works of MacRae and Barnum,[13,14] Franceschetti *et al.*,[6] Epstein,[4] and Kvasnička,[10] the paracondyloid process is confused with the paramastoid process and indicated as such, as if the two terms were synonymous and the anomalies identical. Actually, the anatomic studies of Hayek[8] have shown that the paracondyloid process is a variation which is observed in man, projecting from the paracondyloid surface of the occipital bone and extending in the direction of the transverse process of the atlas, whereas the paramastoid process is a variation observed in many animal species and its usual location is at the posterior border of the jugular foramen. The para-

Fig. 4. A bilateral paracondyloid process.

condyloid process has been compared to the sacralized transverse apophysis of the last lumbar vertebra and, as the latter, it may be uni- or bilateral and may show symmetric or asymmetric development (Fig. 4 and 5*a*). That it orginates from the occipital bone (exo-occipital) is also proved by the presence of pneumatic cells.[1] Tischendorf[20] from an anatomic point of view and Schmidt[16] from a roentgenologic point of view have described a paracondyloid suture situated between the base of the malformation and the occipital bone.

In our material there have been 5 cases of the paracondyloid process—3 unilateral and 2 bilateral. In only 1 case was the malformation present in its pure form; in 3 cases it was associated with assimilation of the atlas and in the remaining case with complex anomalies of the occipitovertebral joint. In 1 case there was an osseous bridge with the atlas, and the paracondyloid suture was never observed. When the paracondyloid process is not fused with the underlying vertebra, it has the shape of a cone, sometimes large, projecting from the occipital bone and with a wide base (Fig. 4). It is bordered by the cortical layer and, in the center, the osseous structure is that of spongy bone; in 1 case pneumatic cells were present (Fig. 5*a*), a phenomenon that appears to be rather rare.[17]

Four patients showed neurologic signs and symptoms that could be attributed to the associated malformations, however, and not to the paracondyloid process

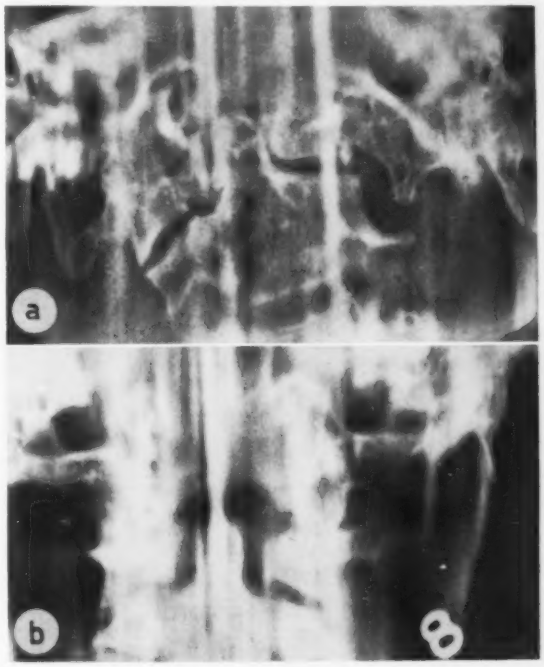

Fig. 5. (*a*) Paracondyloid process on the left. (*b*) Epitransverse process of the atlas fused to the occipital bone.

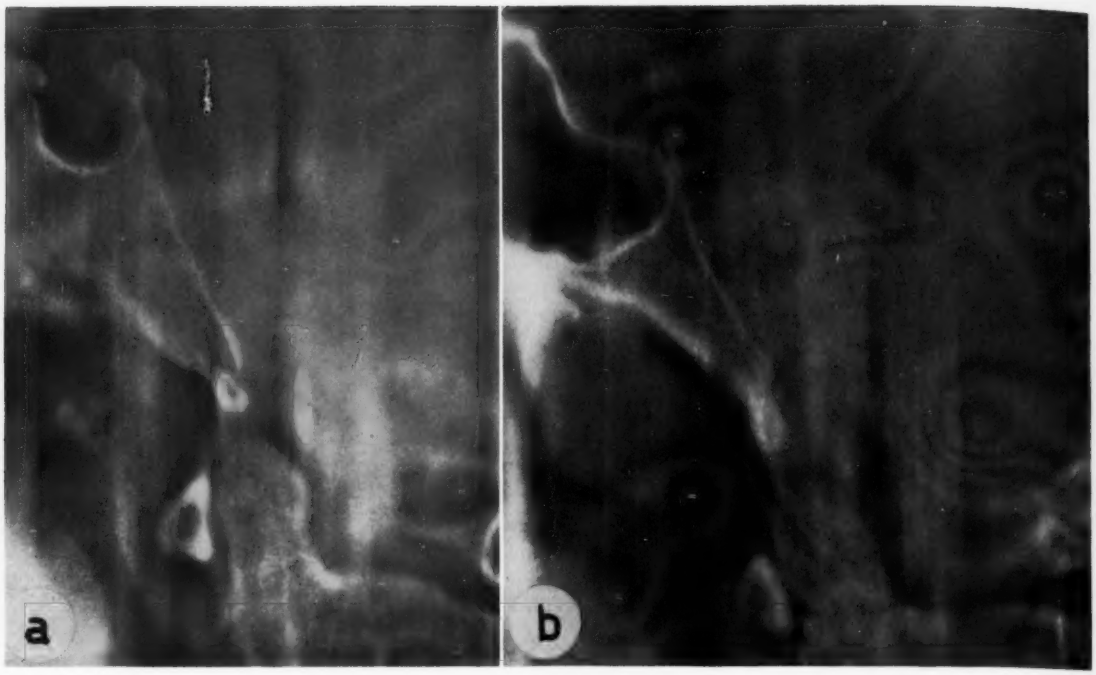

Fig. 6. (*a* and *b*) Basilar processes.

which, in spite of its size and the fusion with the atlas in the fifth patient, caused no disturbances. In Kvasnička's[10] case, on the other hand, the head was fixed in an attitude of flexion.

The paracondyloid process must not be confused with the epitransverse process of the atlas, which is its mirror image (Fig. 5*b*). It is an osseous formation which projects from the transverse process of the atlas with an arch medially concave and extends towards the occipital bone with which it is sometimes fused.[11] The few cases in the roentgenologic literature have been reported by Brocher,[1,2] Viehweger,[22] Schmidt,[16] and Schmidt and Fischer.[17]

BASILAR PROCESSES

Incomplete regression of the lateral parts of the hypochordal anlage may result in the presence of two small, often asymmetric, bone formations disposed along the median line on the inferior surface of the base of the skull at the anterior border of the occipital foramen. Sometimes these formations are in contact with each other, forming a small central canal and simulating a third condyle. Roentgen demonstration of the basilar processes is difficult and they cannot always be recognized with certainty. We were able to diagnose the 2 cases shown in Figure 6, *a* and *b* because of their localization and similarity to the basilar processes seen in the photographs of the dried skull.

ACCESSORY BONE ELEMENTS, SEPARATE OR FUSED TO THE FORAMEN MAGNUM

According to Schumacher,[18] we must regard as a manifestation of the occipital vertebra its ossified fragments which, when imbedded in the ligamentous tissue, appear on roentgenographic examination as isolated foci between the atlas and the occipital bone. We have collected 4 cases of this type and others have been published by Fischer.[5] In the latter's view, the separate bone elements are only partly attributable to the occipital vertebra, because findings due to secondary ossification in the ligaments have been observed.

These accessory formations have the appearance of sesamoid bone and are mostly encountered above the anterior

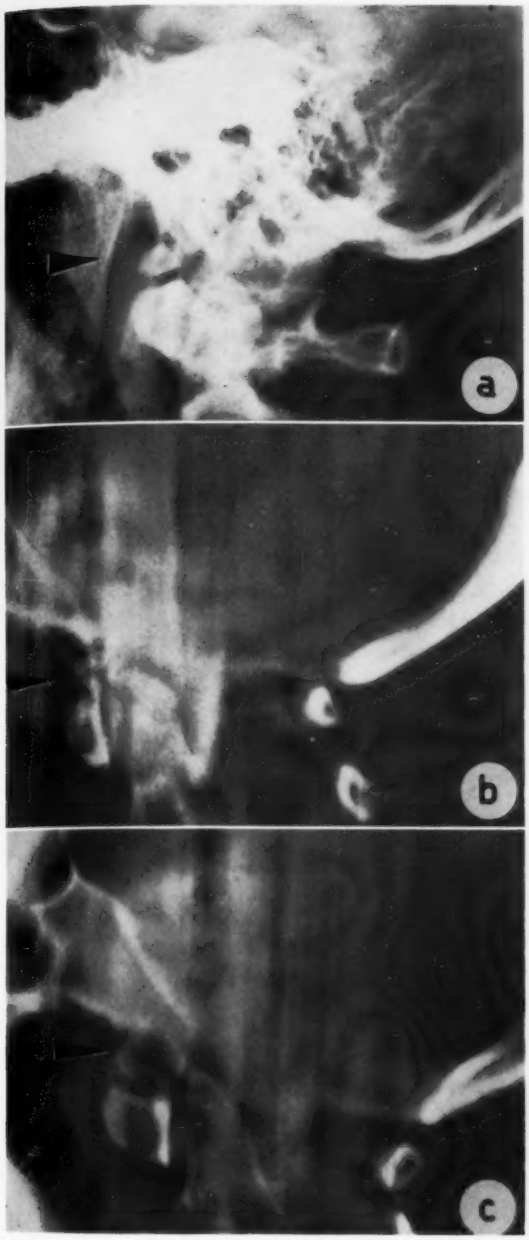

Fig. 7. (*a*, *b* and *c*) Different aspects of accessory bone elements.

arch of the atlas (Fig. 7, *a*, *b* and *c*). This localization renders them easily distinguishable from the ossiculum terminale of Bergmann, a normal anatomic formation which, though not always, is situated on the odontoid process, constituting its tip. In 1 case only, the ossification was localized over the odontoid of the axis, but its semilunar shape clearly pointed to its having originated from a hypochordal arch (Fig. 8). Neither in this patient nor in the other 3 cases were there any physical disturbances ascribable to the malformation.

The neural arch or a transverse process of the pro-atlas may become fused, wholly or in part, with the lower rim of the occipital foramen, which is pinched in and deformed by osseous formations (labia foraminis magni). The case shown in Figure 9 is an example. The patient, a fifty-one year old male, had noted five years previously the appearance of a spastic paraparesis of the lower extremities which progressed slowly and which, after four years, involved the upper extremities as well. When the patient was admitted to the hospital he presented a spastic tetraparesis, more marked in the lower extremities, with mild hypotrophy of the scapulohumeral musculature and slight bilateral superficial hypoesthesia from C_3 to C6. Roentgen examination revealed a fairly complicated malformation. In addi-

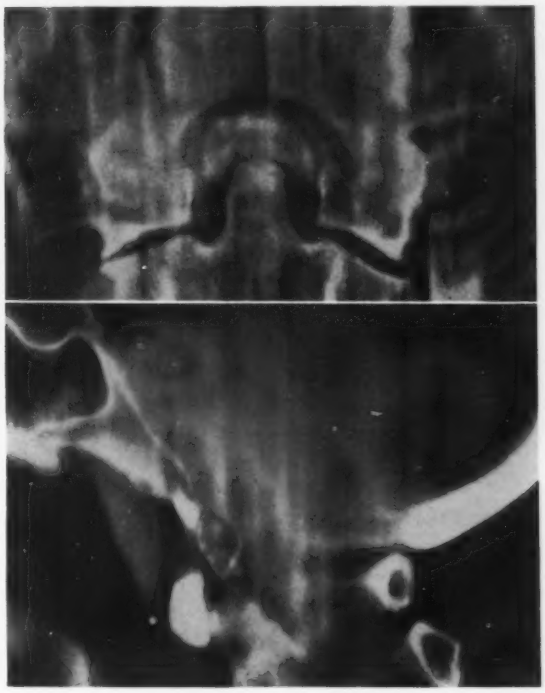

Fig. 8. Accessory bone on the anterior part of the foramen magnum.

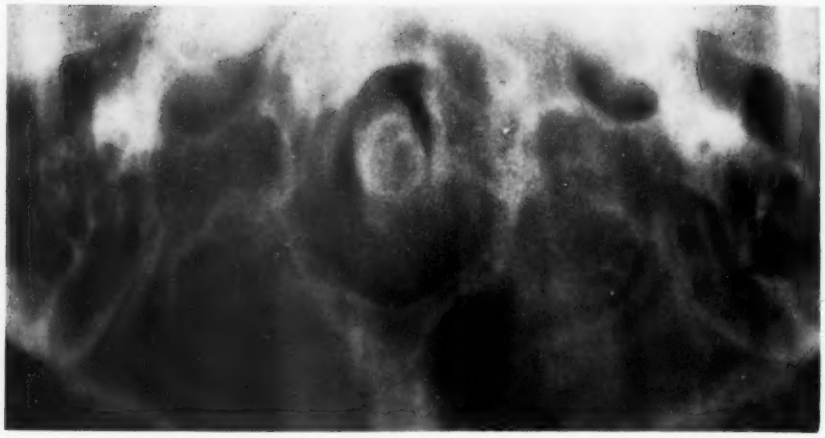

FIG. 9. An axial transverse laminagram showing distortion of the foramen magnum by an anterolateral bony mass on the left and by posterior luxation of the odontoid process.

tion to the asymmetric basilar impression with platybasia and posterior dislocation of the odontoid process of the axis, two simultaneous varieties of manifestation of the occipital vertebra were present, *viz.*, a paracondyloid process and a bony mass distorting the occipital foramen (Fig. 9). In view of the complex character of the malformation encountered, it was impossible to determine the part each of these played in provoking the clinical symptomatology exhibited by the patient. In bilateral cases, *e.g.*, the case reported by Hadley,[7] the occipital foramen showed a deformation in the shape of a bicycle saddle.

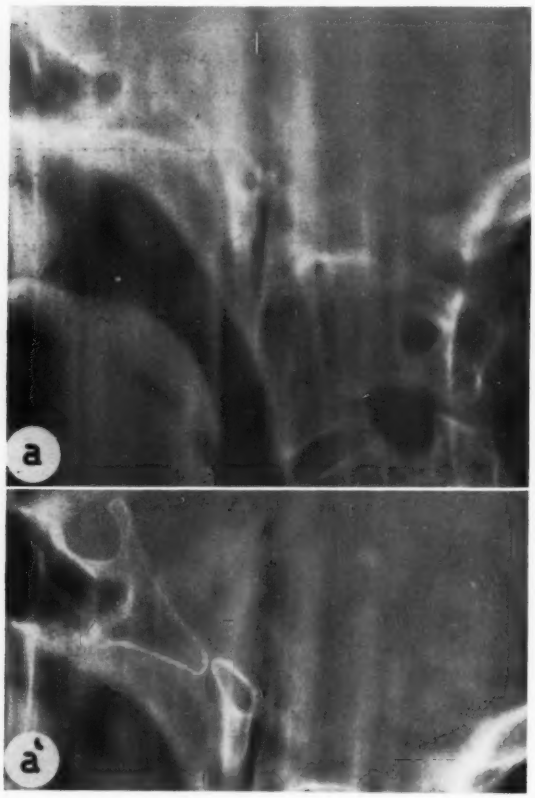

FIG. 10. (*a*) Laminagram with paramedian section showing lateral segmentation of the basiocciput. This fissure is of the same type as 1 in Figure 11. (*a'*) A retouched reproduction from *a*.

TRANSVERSE FISSURES OF THE BASILAR PROCESS OF THE OCCIPITAL BONE

These constitute another aspect of manifestation of the occipital vertebra. Very rarely, the fissure may be complete, in which case it borders an arch which Chiarugi has called the "pre-basioccipital arch." More often, it is partial, uni- or bilateral.[11,15–17] Neither variety has clinical significance. As far as we could determine, roentgenologic documentation is available only for a case described by Schmidt and Fischer[16,17] and one by List[12] which showed a unilateral fissure of the basiocciput and assimilation of the atlas. Our case (Fig. 10, *a* and *a'*; and 12*c*) is identical with the latter even as regards the associated malformation. Laminagraphy of the basiocciput performed in a lateral plane revealed a distinct interruption.

Figure 11 illustrates, by line drawings

superimposed on a laminagram, the various types of fissures observed in photographs of anatomic specimens.

DISCUSSION

In contrast to previous assumptions, and with a frequency surpassing that mentioned in the literature, it has been our experience that roentgenologic investigations may reveal the bone anomalies of the manifestation of the occipital vertebra.

These are anomalies of considerable interest anatomically and, from the phylogenic point of view, they have the significance of a regressive trait. There is an apparent contradiction, explained by embryology, in that they have morphologic characteristics of a vertebral type identical to those of the atlas, although they belong to the skull (spondylocranium), as is clearly evidenced by their fusion and assimilation with the occipital bone.

These malformations are rare. Sauser[15] has observed only 5 cases among 1,119 skulls in an ossuary (0.44 per cent); we have found 19 such cases in 4,000 patients (0.47 per cent). The relative frequency of our observations is, therefore, the same as that of Sauser,[15] which can be explained by the fact that the limitations of a roentgen examination are offset by the demonstration of the isolated ossifications, which become lost in the dried skull.

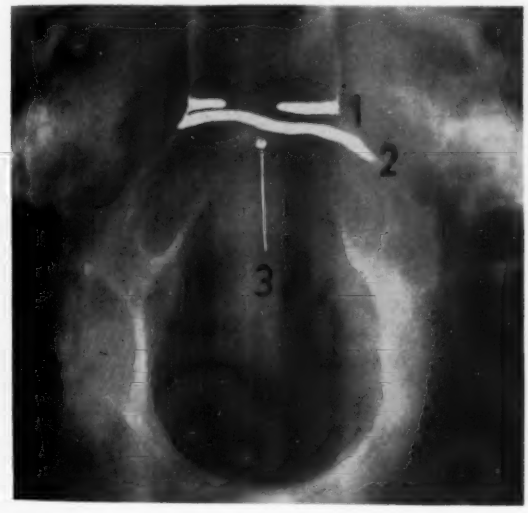

FIG. 11. Types of fissures schematically indicated by superimposed lines: (1) lateral segmentation, (2) complete segmentation of the basiocciput due to lack of assimilation of the most hypoglossal sclerotome, (3) pre-basioccipital arch.

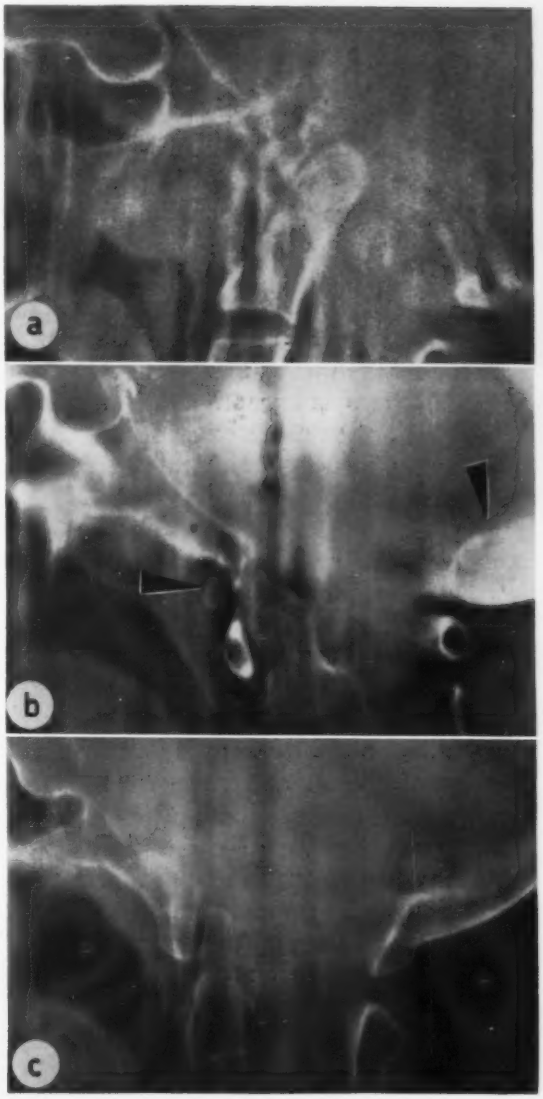

FIG. 12. Laminagrams demonstrating (*a*) dysplasia of the basiocciput, (*b*) manifestation of the occipital vertebra with deformity of the anterior and posterior parts of the foramen magnum, and (*c*) atlanto-occipital fusion with the second and third cervical block vertebrae. (Same case as in Figure 10, *a* and *a'*.)

Most of the time manifestations of the occipital vertebra have no clinical significance. There are rare exceptions: the third condyle, when well-developed, may limit

the mobility of the head; the paracondyloid process, fusing with the transverse process of the atlas, may force the head into a continuous attitude of flexion (caput obstipum) and, most importantly, the abnormal osseous formations which originate from the arches or from the transverse apophyses of the pro-atlas, when fused with the base, will narrow the occipital foramen and may provoke neurologic symptomatology of a compressive type.

The roentgen demonstration of these particular cases is exceptional, but it is to be expected that more cases will be diagnosed in the future, mainly as the result of the application of axial transverse laminagraphy. At the present time, so far as we have been able to ascertain, the only cases that have been diagnosed in the living patient have been the one reported by Hadley[7] (in which operation was carried out), the case of Brocher and Klein[3] (twelve year old boy who presented an incomplete form of mandibulofacial dysostosis [syndrome of Treacher-Collins]), and the case reported in the present communication.

The manifestation of the occipital vertebra may be present in its pure form or in association with other local malformations. In our group of cases, the pure form was encountered more often (11 cases) than the associated form (8 cases). In this connection we wish to call attention to the high frequency of the paracondyloid process in cases of assimilation of the atlas. The 5 cases out of 25 reported by McRae and Barnum[14] show the same proportional frequency as the 3 out of 14 cases in our material; the causes of this curious association are not clear.

One patient, in addition to malformations of another nature, presented a paracondyloid process and a bony mass distorting the occipital foramen, *viz.*, two variations of manifestation of the occipital vertebra.

The study of this malformation has, furthermore, demonstrated the possibility of reconstructing roentgenologically even to details, the complex evolutive developments of the occipitovertebral region and of compiling a sequentially progressive list of malformations: severe dysplasia of the basiocciput, manifestation of the occipital vertebra, and assimilation of the atlas (Fig. 12, *a*, *b* and *c*). The links between these stages are intermediate forms.

CONCLUSIONS

The occipital vertebra is a rare malformation which, although known to anatomists, is not very familiar to radiologists.

Though the anomaly is normally asymptomatic, it may, alone or in combination with other local malformations, provide neurologic signs and symptoms.

Roentgen examination of the craniovertebral junction in a consecutive series of 4,000 patients has revealed manifestation of the occipital vertebra in 19 cases (0.47 per cent).

The most common of these malformations is the third condyle, followed in descending order of frequency by the paracondyloid process, isolated ossification between the atlas and the occipital bone, basilar processes, fissures of the basiocciput, and a bony mass fused with the occipital foramen. The last mentioned variant appears to be the most dangerous form because it may lead to direct compression of the spinal cord.

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ANATOMICO-ROENTGENOGRAPHIC STUDIES OF THE POSTERIOR SPINAL ARTICULATIONS

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KELLER,[2,3] from his studies of the apophyseal articulations, has reported that local back pain arises from the posterior spinal joints. The present study was undertaken for the purpose of coordinating the roentgenographic appearance with the anatomic condition of these structures.

ANATOMY AND FUNCTION

The lower element of the joint, *i.e.*, the superior articular process, is concave or partially cylindrical in the lumbar region, while the inferior articular process is biconcave (Fig. 2). Since the surfaces are not completely in contact, meniscus-like tabs of synovial tissue project inward from the joint margins. These lie partially between the articular surfaces. Some of them are thick and fleshy; others are thin, fibrous and ragged. They are made up of fibrous tissue, fibrocartilage, fat cells and many blood vessels (Fig. 2, 5 and 6).

Medially and anteriorly, the joint capsule is formed by a lateral continuation of the tough ligamentum flavum. Posteriorly, the capsule is much thinner and is loosely attached, not to the margins of the joints, but reflected around to the outer surfaces of the articular processes. The articular cartilage likewise may extend well beyond the limits of bony contact. The joint space

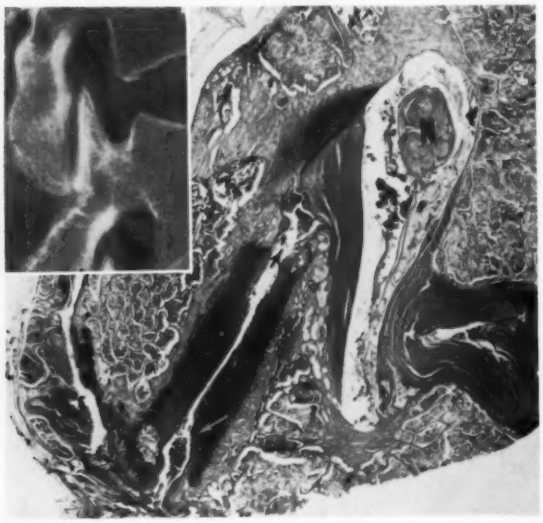


FIG. 1. Sagittal section (×2) of a normal fourth posterior lumbar articulation in a thirty-seven year old male. The capsule above, formed from the ligamentum flavum, separates the joint from the normal sized intervertebral foramen on the right. Note that the diameter of the nerve (N), which in the lumbar region occupies the upper portion of the foramen, is no more than one-fifth the size of the opening which normally transmits it. The dark articular cartilage on both sides shows a normal thickness. Tabs of synovial tissue are visualized above and below (arrows). Note the customary weakness of the capsule at the bottom. (The insert is a roentgenogram of this joint.)

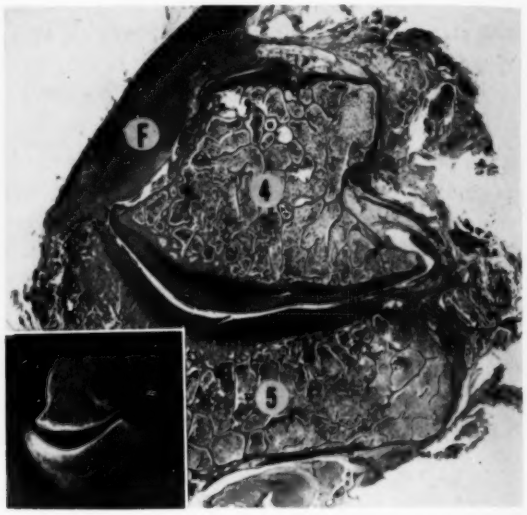


FIG. 2. Transverse section (×3) of the fourth posterior articulation in an eighty-one year old male. The articular surface of the lower element from the fifth vertebra inferiorly and anteriorly is concave and covered with hyaline cartilage (5). This opposes the convex cartilaginous surface of the upper element (4). The ligamentum flavum (F) constitutes the capsule on the medial aspect of the joint. The arrows indicate the reflection of the capsule from the bone on each side. Note the laxity of the capsule and the synovial tissue lying within it. (The insert is a roentgenogram of the specimen before section.)

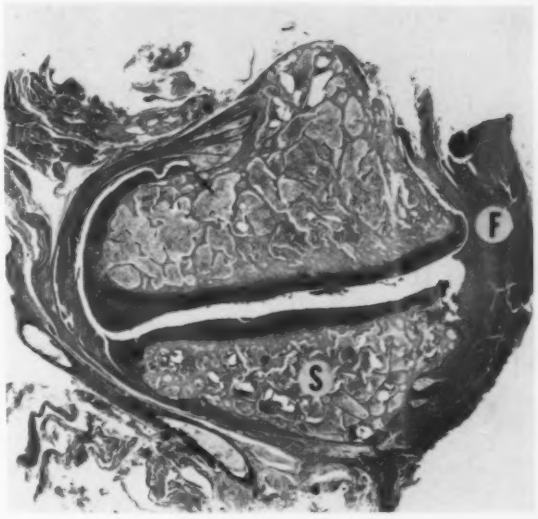


Fig. 3. Transverse section (×3) of the fifth posterior articulation from a seventy-three year old male. At the top is the triangular section from the tip of the fifth inferior articular process; the articular process from the sacrum is visualized below (S). A cushion of cartilage is reflected around the right articular margin sustaining the sidewise thrust of the joint by its pressure against the ligamentum flavum at F, which here constitutes the anteromedial portion of the capsule. On the left, the side thrust is opposed by the inside of the capsule where the cartilage is reflected completely around to the posterior surface of the articular process (arrow). It will be seen that in the lower lumbar region the posterior joint offers considerable laxity of movement in all directions and that the cartilage articulates with ligamentous structures.

may thus be actually extended well around to the posterior surface of the articular process. Laxity of the capsule posteriorly and inferiorly allows considerable range of movement in different directions. The cushions of cartilage along the sides of the articular processes serve as bearing points against the inside of the capsule of the joint. This enables it to resist the sideways thrust of the upper element of the articulation (Fig. 3 and 4).

From a study of cadaver material as well as routine roentgenograms, it is obvious that these joints permit various types of gliding movement. Upon anterior flexion the upper element glides upward and forward upon the lower element, the articular surfaces separate at the lower margins of the joint and the foramen opening becomes larger. Only a portion of the articular surfaces may remain in contact. Upon backward bending the joint surfaces telescope into each other and the foramina become smaller. Upon rotation, the articular sur-

Fig. 4. Dissected fifth lumbar capsule from a seventy-three year old male showing how the structure is attached well beyond the articular margin. The synovial tab which projects between the joint surfaces like a meniscus also can be seen. The overhang of the joint margin is much greater at the lumbosacral level than it is above. On the opposite side, this subject presented a double fat pad with smooth surfaces which acted as a cushion between the tip of the fifth right inferior articular process and the neural arch of the sacrum.

Fig. 5. The third right posterior articulation from a sixty-nine year old male laid open to show the articular surfaces and the synovial menisci. The capsule was detached from the posterior surface of the upper element at X and was retracted downward. It bears a long, thin, ragged, fibrous synovial tab or meniscus (black arrows). A thick fleshy meniscus of fatty tissue is seen attached to the upper portion of the capsule but displaced slightly downward (white arrow). The thick meniscus was present above because the upper margins of the joint were well separated. The darker, concave, lower element of the joint beneath the fat pad was roughened by early degenerative change. Note also some roughening of the posterior edge of the upper element just to the right of X.

faces press together on one side and tend to pull apart on the other. With lateral flexion, balanced gliding movements occur with the upper joint margins separating on the convex side of the curve and imbrication or telescoping occurring on the concave side.

Sections of the joint capsule studied by special staining methods visualized a rich supply of sensory nerves (Fig. 7). May we not assume the possibility of a pinching of the soft tissue structures of the joint as a cause of the "sudden catch" type of low back pain? Clinically, this is characterized by the patient's inability to completely straighten up after bending forward.

DEGENERATION

Normally, the articular surfaces are covered by smooth glistening hyaline

Fig. 6. Photomicrograph (×4) showing a large synovial tab (arrow) projecting upward between the articular surfaces. This originates from the inferior, loose portion of the capsule. The structure in this instance is largely composed of fatty tissue. The articular cartilage on the right side is partially worn down but the hyaline cartilage is of normal thickness on the left. This synovial tab might conceivably become pinched between the bony structures.

cartilage. Early degenerative changes first appear as a velvety texture overlying the articular surface (Fig. 5). Later, the cartilage becomes fibrillated and fragmented and finally disappears entirely in some parts of the joint. Roentgenograms of these joints resemble those of some patients complaining of local back pain (Fig. 8).

IMBRICATION, IMPINGEMENT, BUMPER FORMATION, EBURNATION AND EROSION

Lordosis, tipping of the sacrum, or thinning of the intervertebral disk results in a telescoping or imbrication of the posterior articulations (Fig. 9, *A* and *B*). The articular surfaces are no longer exactly opposite each other. A pulling stress is placed upon the capsule. The anterior inferior element of the joint, by moving upward and forward, may encroach upon the foramen, even to the extent of pressing upon and distorting the shape of the nerve root.

If imbrication is excessive, a painful bony impingement may occur between the tips of the articular processes and the adjacent portions of the neural arch (Fig. 10). A fibrocartilage pad or bumper develops on the tip of the impinging articular process at the point of recurrent pressure (Fig. 11, *A* and *B*) and a sclerosis or eburnation of the underlying bony structure results (Fig. 12, *A*, *B* and *C*). This tends to counter the localized pressure stress. With continued pressure at one point, however, the condition progresses to actual erosion of the underlying bony tissue.

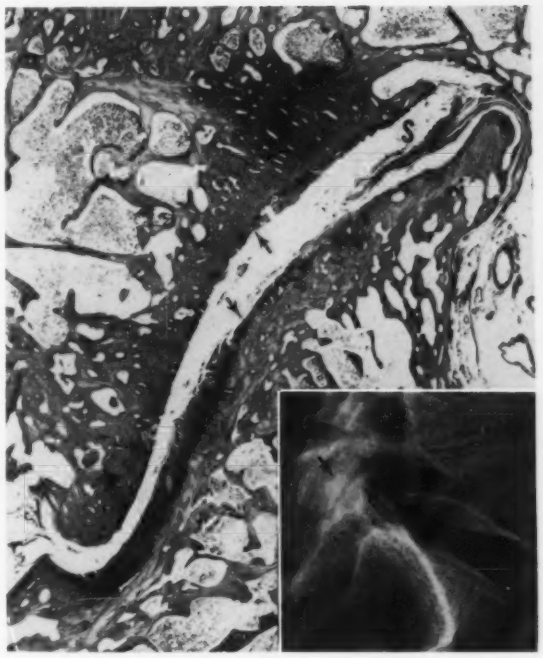


Fig. 8. Longitudinal section (×5) of a degenerated fifth posterior joint from a seventy-five year old male. This shows the synovial element (S) projecting into the upper margin of the joint space. The cartilage is completely absent from the upper element of the joint and there is advanced cartilage degeneration with thinning and fibrillation below (white arrow). (The insert is a roentgenogram of the specimen before section showing the narrowed joint space.) Sclerosis of the bone adjacent to this degenerated joint is well seen both on the section and the roentgenogram (black arrows).

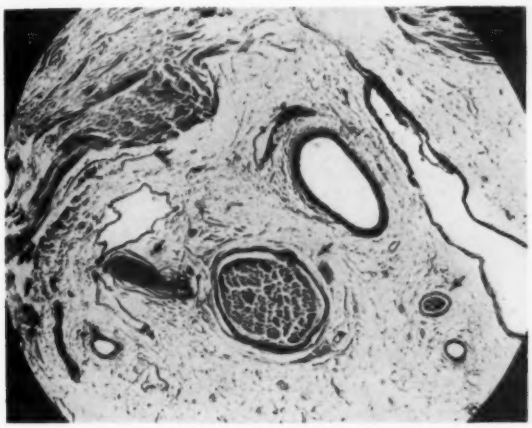


Fig. 7. Section (×30) from the posterior joint capsule showing at least three sensory nerves (arrows). In the center is a large mixed nerve having both myelinated and unmyelinated fibers. Some of the latter are seen branching off from its upper surface. Smaller nerves appear on the right and left. The former is seen in cross section, while the dark oval shadow on the left is an obliquely sectioned sensory nerve. (This section was stained by a modified Bodian and Ungewitter silver nerve stain.)

COMMENT

Harris and Macnab[1] have discussed the relationship of symptoms to posterior joint changes. Assuming, as Keller[2,3] has

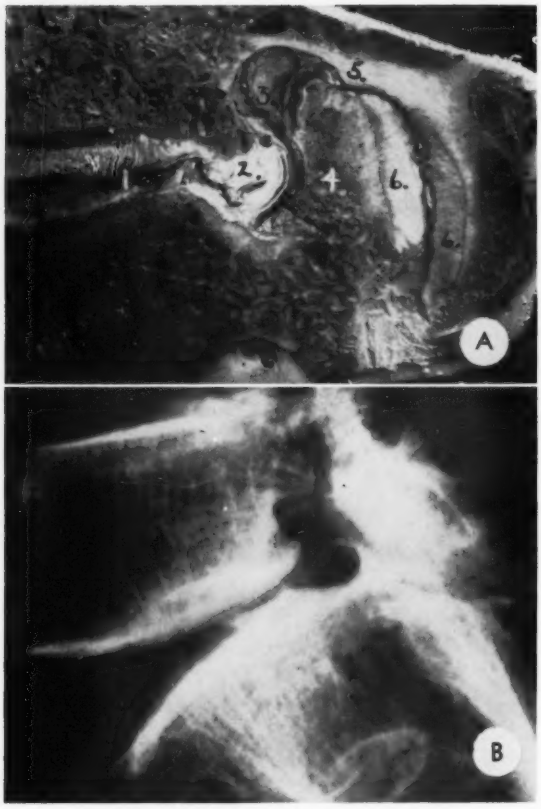


Fig. 9. (*A*) Section at the lumbosacral level from an eighty-six year old male showing imbrication of the posterior articulation. The articular cartilages (6) do not register exactly opposite each other. The articular process of the sacrum (4) has telescoped upward, impinging against the eburnated fifth pedicle at 5 and distorting the nerve root (3). (*B*) Roentgenogram showing the wedge-shaped lumbosacral disk. There is some tipping of the sacrum on a transverse axis. The fifth vertebra is displaced backward upon the sacrum. The disk (1 in *A*) is thinned posteriorly and its annulus fibrosus (2 in *A*) has become displaced backward and encroaches upon the lower part of the intervetebral foramen.

Fig. 10. Section of the fourth right posterior articulation of a seventy-three year old female. The insert is a roentgenogram showing impingement of the tip of the fourth inferior articular process (4) against the isthmus of the fifth neural arch. This has produced erosion with the formation of a compensatory eburnation of bony tissue at the point of pressure (X). The tips of the articular processes subjected to intermittent pressure usually develop fibrocartilage bumpers at points where the normal hyaline articular cartilage does not occur. Such bumpers are noted in the section as indicated by the arrows. Richly vascular synovial tabs are seen above and below at S. There is advanced cartilage degeneration with fibrillation and complete loss of cartilage in the upper portion of the joint.

→

Fig. 12. (*A*, *B* and *C*) Scoliosis in an eighty-four year old female incidental to wedging of the lumbosacral disk and the fifth vertebral body (5). There is telescoping, impingement, eburnation and erosion of the fifth posterior articulation on the right side (R, arrow). Compare with the normal condition of its fellow on the left (L).

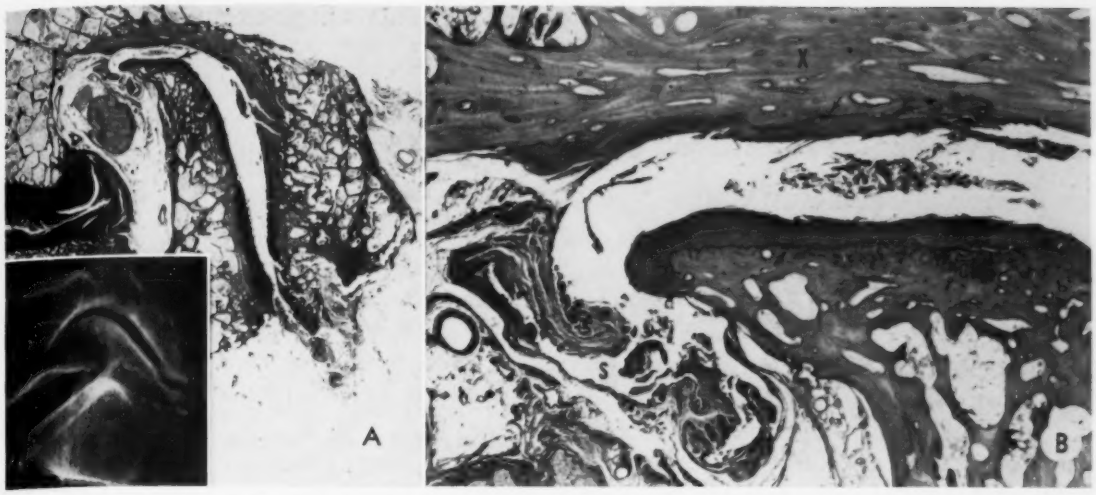
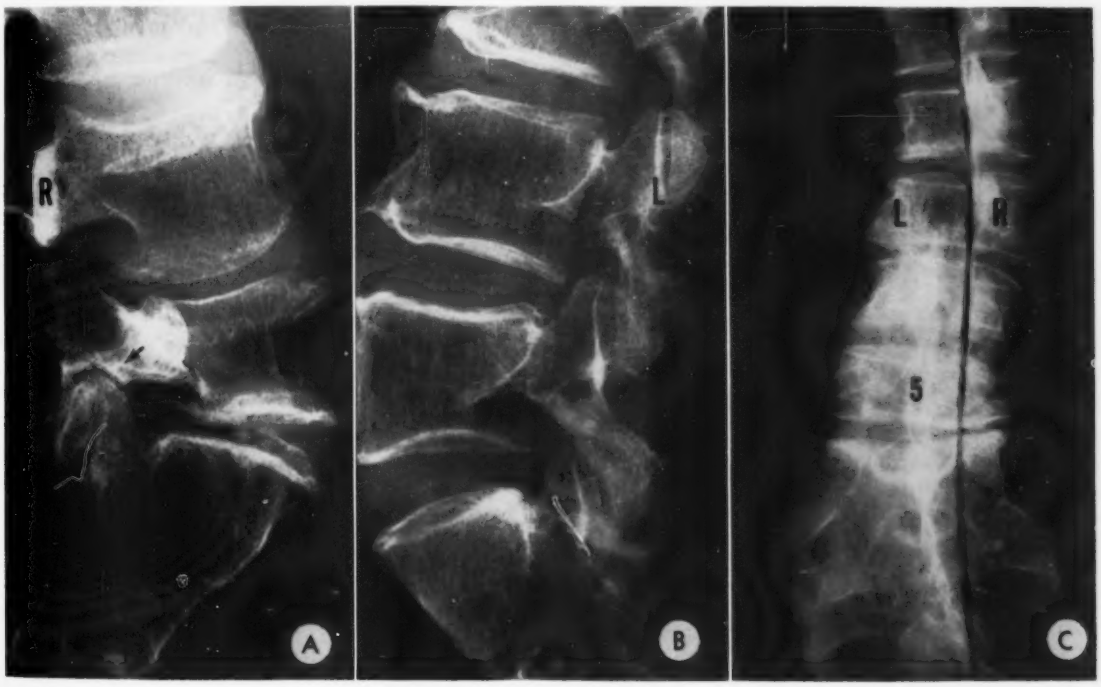


Fig. 11. (*A*) Longitudinal section (×1½) of the fifth lumbar articulation of a seventy-nine year old female. Imbrication and bony spur formation has progressed until extensive impingement has occurred beneath the fifth pedicle with eburnation of the latter. A fibrocartilage bumper at the point of contact has developed on the tip of the sacral articular process (arrow). There is nearly complete loss of articular cartilage below this point. The intervertebral foramen on the left is not involved. Two elements of the motor nerve root are seen slightly above and to the left of the much larger, darker sensory nerve root. (The insert is a roentgenogram showing sclerotic change in the bony structure of the joint.) (*B*) Photomicrograph (×18) of the tip of the sacral articular process showing the fibrocartilage bumper (B), a completely anomalous structure which has developed at the point of pressure. This is not the hyaline type of cartilage which normally covers the articular surfaces, as seen in Figure 6. The opposing sclerotic bone of the pedicle (X) is also protected by a slightly thinner coat of fibrocartilage. These cartilage bumpers are separated from the underlying bone by a narrow zone of darkly staining active cells (arrows). Evidence of new bone growth is seen in the spur formation beneath the bumper and just to the left of the lowermost arrow. Tabs of highly vascularized synovial tissue (S) are noted between the joint and the intervertebral foramen. This section was taken somewhat lateral to the ligamentum flavum, so the capsule between the joint and the intervertebral foramen is thin and delicate as compared with that noted in Figure 1.



stated, that local back pain arises in the posterior joint structures, a coordination of the roentgenograms with the anatomic studies should contribute something toward an understanding of this problem.

SUMMARY

The posterior spinal articulations are gliding joints with a considerable range of movement. There are menisci of synovial tissue projecting inward between the bony articular surfaces. The joint capsule is rich in sensory nerve fibers. The possibility that pinching of the soft tissues causes the "sudden catch" type of low back pain is proposed. Various kinds of joint destruction, with imbrication, impingement, bumper formation, eburnation and even erosion, probably play a part in the production of local back pain. The 45° stereoscopic projection is recommended to best visualize the joints in roentgenography of the lumbar region.

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Assistance was given in these studies by the Departments of Anatomy and Photography, New York State School of Medicine, Syracuse, New York.

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A VERTICAL FRACTURE OF THE FIFTH CERVICAL VERTEBRA WITHOUT NEUROLOGIC SYMPTOMS

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VERTICAL fractures of the cervical vertebrae are almost without exception accompanied by quadriplegia or paraplegia.

This article reports a case of a vertical fracture of C_5 with no neurologic symptoms.

REPORT OF A CASE

A twenty-seven year old male was admitted for observation following a history of an automobile accident; he had suffered momentary unconsciousness when his small foreign car had been struck from the rear by a truck.

Physical examination on admission was completely negative, except for a hematoma in the occipital region.

Roentgen examination showed evidence of a vertical fracture of C_5 with a slight compression of the anterior-superior portion. No other abnormality of the cervical spine was noted (Fig. 1, *A* and *B*).

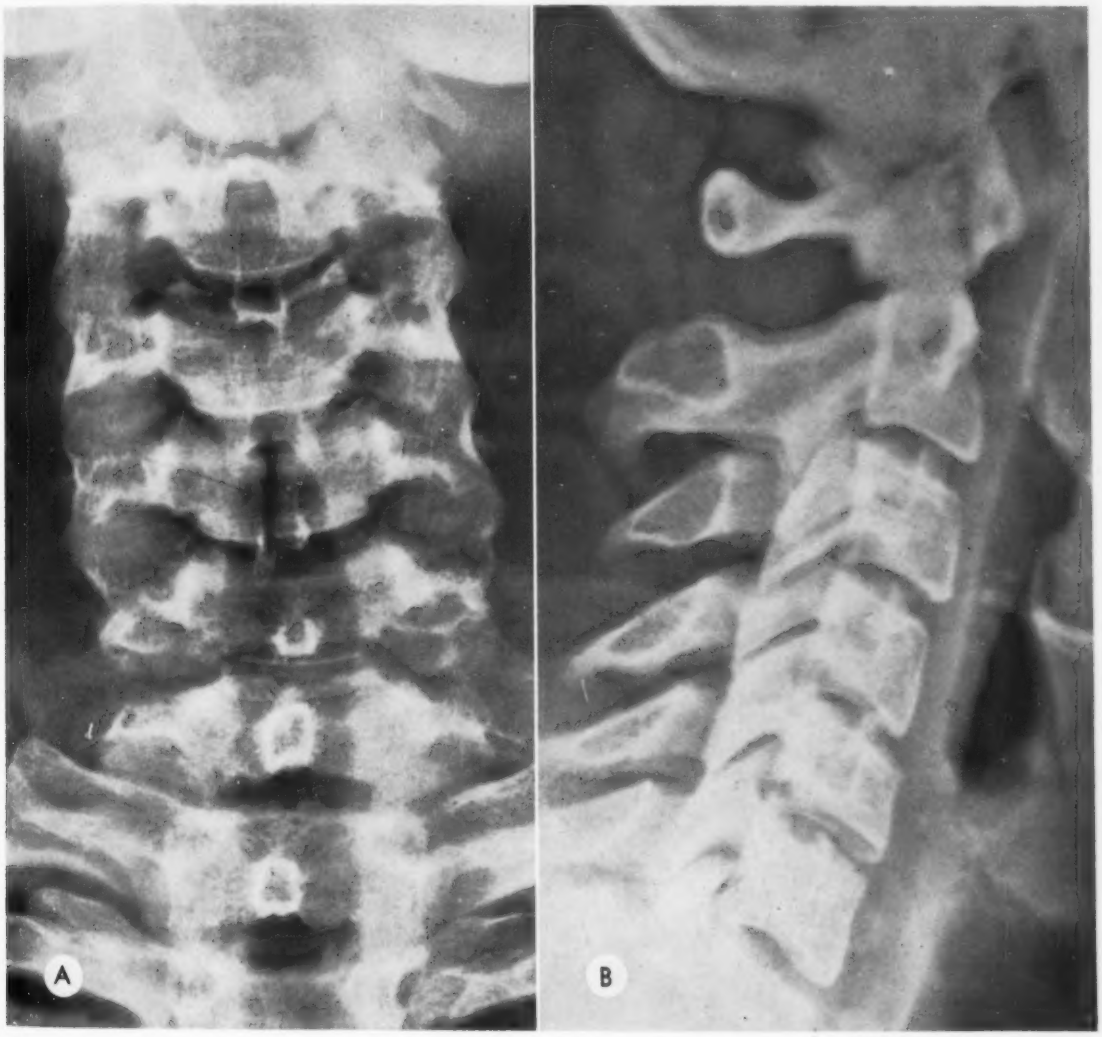
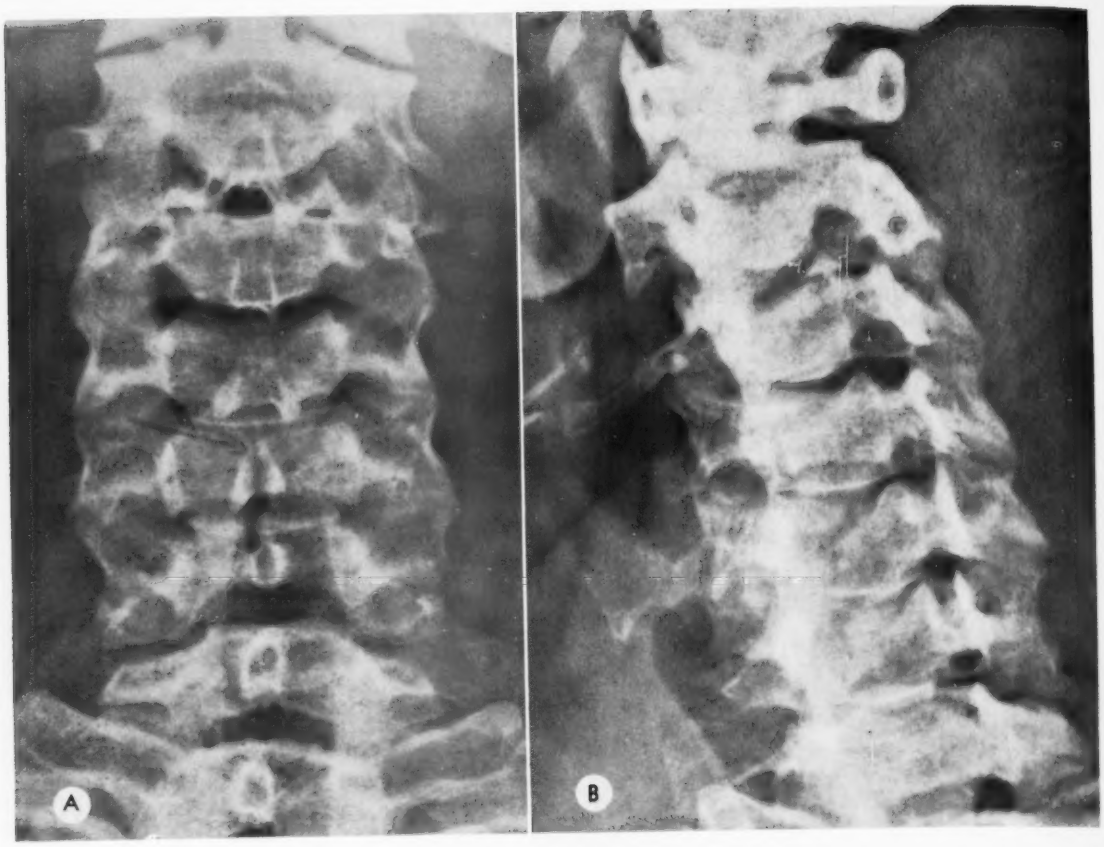


FIG. 1. (*A*) Roentgenogram showing vertical fracture of C_5. Note the downward displacement of the left half of the vertebral body. The radiolucent line is seen within the air column. (*B*) Note that there is some compression of the anterior height of C_5.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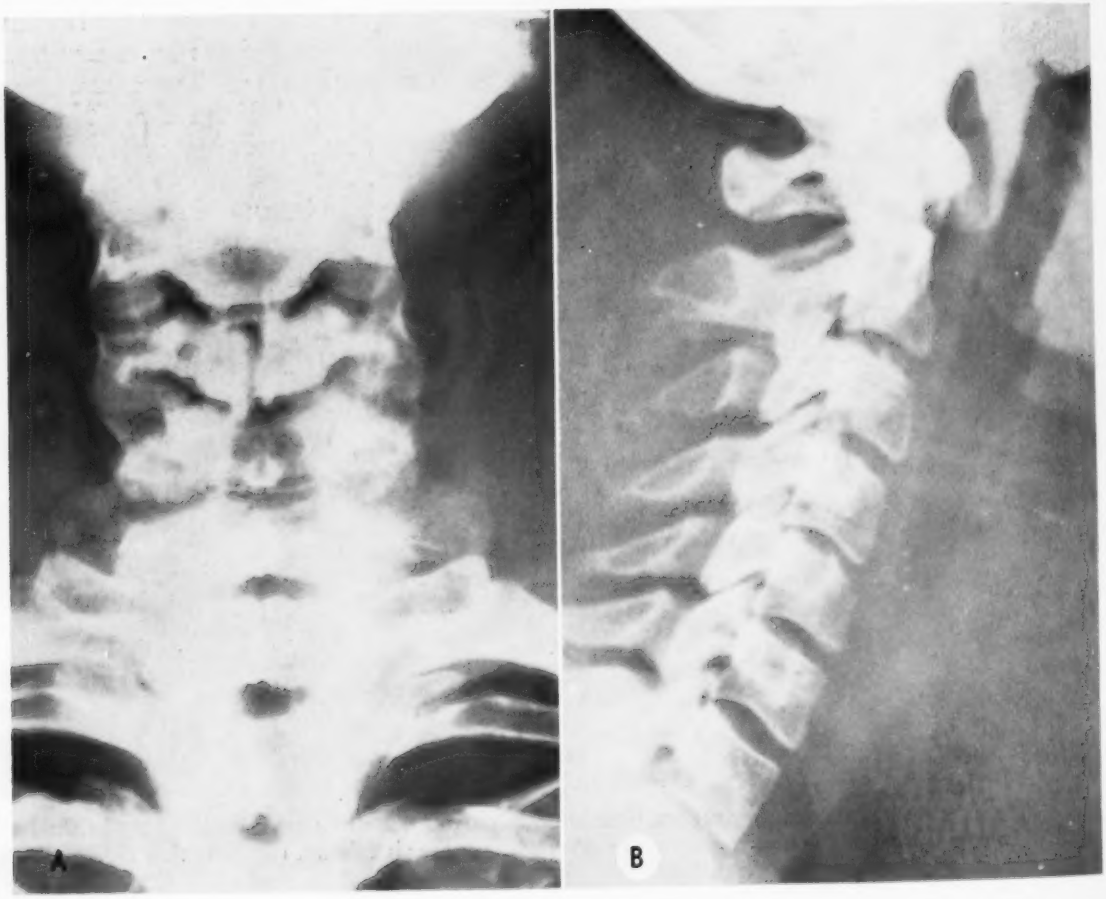
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Fig. 2. (*A* and *B*) Roentgenograms taken one month after Figure 1.

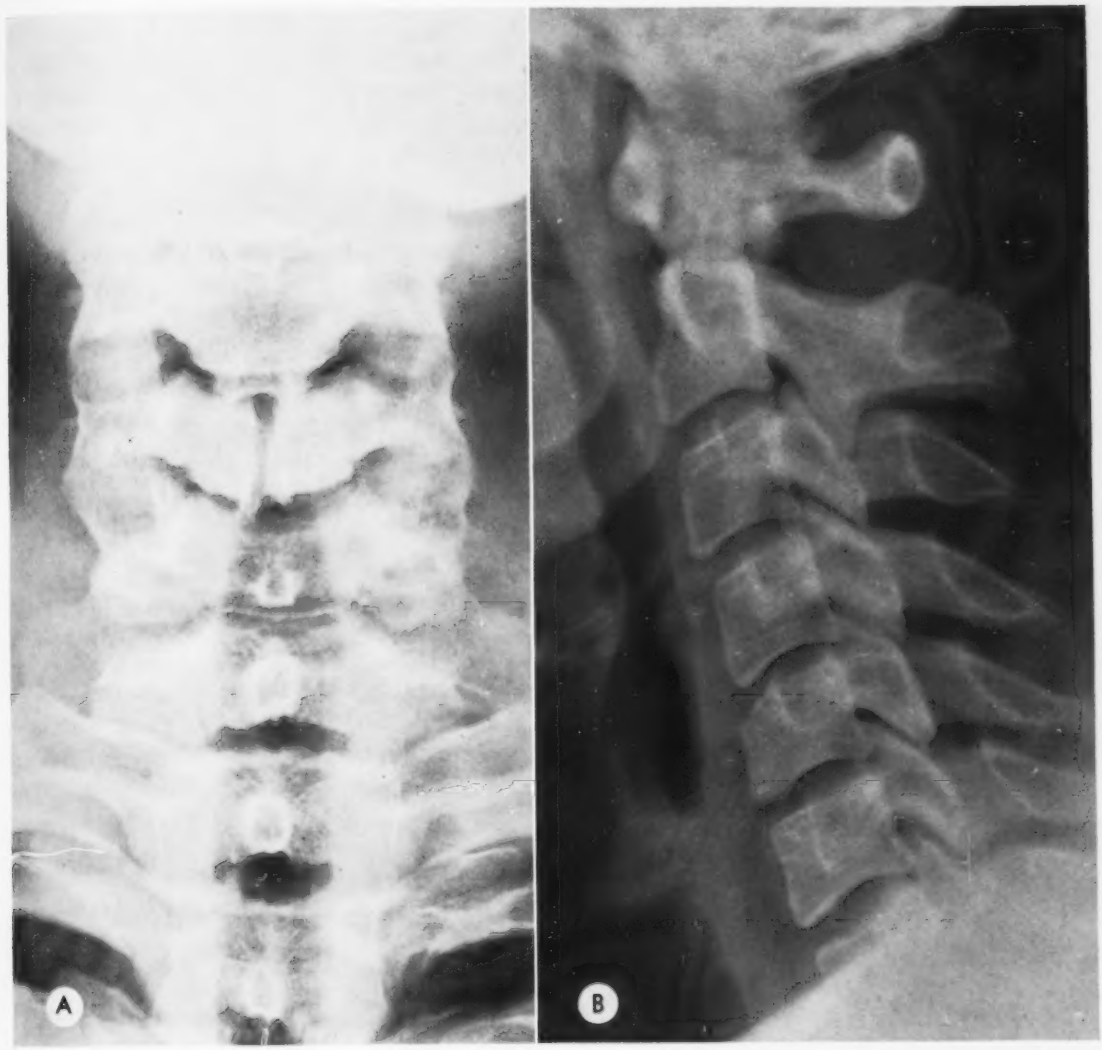

Fig. 4. Roentgenograms taken almost three months after Figure 1. (*A*) There is further decrease in the anterior height of C5. (*B*) A slight amount of bony bridging is present.

Fig. 3. (*A* and *B*) Roentgenograms taken approximately two months after Figure 1.

The patient was placed in cervical halter traction, which was followed later by use of a cervical brace. Periodic roentgenograms were taken (Fig. 2, *A* and *B*; and 3, *A* and *B*).

When he was last seen eighty-five days after admission, he still had no neurologic symptoms. The roentgenograms made at that time showed further decrease in the anterior height of C_5 (Fig. 4*A*). Some bony bridging of the fracture fragments was also present (Fig. 4*B*).

COMMENT

The patient does not remember receiving a blow to the occipital region. However, there had been a large amount of luggage stacked in the back of his small car and one of these pieces may have become a missile. The patient's neck probably went through the same motions as are carried out in a "whiplash" injury. It would be of interest to know what part the blow to the occipital region contributed to the end result.

The possibilities of the radiolucent line in the vertebra being air in the trachea, a failure of union of the vertebral body or a spina bifida occulta were considered. The first was ruled out because of the roentgen appearance and the downward displacement of the left half of the vertebral body. The last two were eliminated on the basis of a routine army induction chest roentgenogram which had been taken approximately two years prior to the accident and on which C_5 could be fairly well visualized and appeared to be normal.

REVIEW

A review of American and English literature for the past twenty years failed to produce a documented case of a vertical fracture and without neurologic symptoms, although mention of such a case was made by Ellis.[1] The possible mechanisms of vertical fractures have been discussed thoroughly by Richman and Friedman.[2]

SUMMARY

A case of a vertical fracture of a cervical vertebra with no neurologic symptoms has been presented.

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The author wishes to thank Doctors S. P. Peartree, W. W. Jurgensen and R. McDonald of Omaha, Nebraska, for their consultation.

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LAMINAGRAPHY OF THE TEMPORAL BONE*

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ROENTGEN examination is a valuable aid in diagnosing diseases of the ear associated with changes in the osseous structure. The roentgenograms must be taken with the patient in positions designed to reveal abnormalities in the area most frequently involved in chronic ear disease, *viz.*, the attic-aditus-antrum area. In projections such as Schüller's, Stenvers', Runström's, Chaussé's, etc. it is possible to demonstrate major or minor parts of the inner aspect of the temporal bone. Better results are sometimes obtained by supplementing the examination with stereoscopic views.

With plain roentgenography of the temporal bone, it is impossible to eliminate visualization of the surrounding structures which will blur the delicate details in the anatomy of the inner ear. The only way of visualizing the inner aspect of the temporal bone without also reproducing the interfering structures on the film is by laminagraphy.

Numerous laminagraphic studies of the temporal bone have been reported since Zeidses des Plantes[10] and Bartelink[1] employed a curvilinear laminagraph in a few cases. Most investigators, however, have used linear laminagraphy, although, theoretically, laminagraphy with polycyclic movement gives the most exact laminagraphic plane with blurring of the surroundings, in contradistinction to rectilinear laminagraphy, which only blurs perpendicular to the tube movements.

EQUIPMENT

In 1950, Sans and Porcher[7] designed a laminagraph called the Polytome, which makes it possible to use four types of obscuring movements. The trajectories are linear, circular, elliptic, and "hypocycloidale." Table 1 shows the various specifications of the Polytome.

The roentgen-ray table may be tilted into any position between horizontal and vertical. The distance between the focus and the plane of cut is constant, 110 cm. The distance from the film to the plane of the cut is, however, variable, since two cassette holders may be used, one placed at the minimum distance from the plane of cut (magnification 1.3) and the other at the maximum (magnification 1.6). The tube is furnished with a fine focus (0.3×0.3 mm.) and a 1×1 mm. focus. The cassette holder at the minimum distance from the plane of cut is provided with a so-called "rotating grid." The cassette holder with the magnification of 1.6 can be inclined.

Owing to these advantages, we chose the Polytome for laminagraphy of the temporal bone, selecting the polycyclic trajectory, named "hypocycloidale" by the manufacturers.

TECHNIQUE AND FINDINGS

Various projections have been used for laminagraphy of the temporal bone. James[4] has described an anteroposterior and an oblique lateral view; Mündnich and Frey[6] reported no less than 5 different projections, *viz.*, an anteroposterior, a lateral, a longitudinal and an axial view of the pyramid, and an axial view of the head. According to our experience with about 250 laminagraphies of the ear, the anteroposterior and lateral projections of the temporal bone are the most useful. In our opinion, these two views give, in most cases, sufficient detail of the temporal bone. Another reason for restricting laminagraphy to one or two projections is to reduce the roentgen-ray dose, as each projection requires six to ten cuts.

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Aided by a grant from the King Christian X Foundation.

TABLE I

SPECIFICATIONS OF THE POLYTOME*

Angle of Cut	Linear† Trajectory	CD	Elliptic Trajectory	CD	Circular Trajectory	CD	Hypocycloidal Trajectory	CD	SC
29°	56 cm.	1			176 cm.	3.1			2.3 mm.
36°	69 cm.	1			217 cm.	3.1			1.8 mm.
40°	77 cm.	1	186 cm.	2.4					1.6 mm.
48°	92 cm.	1					451 cm.	4.9	1.3 mm.
60°	115 cm.	1							1.0 mm.

* The table lists, for each angle of cut, the various trajectories, the length of each at the level of the roentgen-ray tube focus, and the minimum coefficient of distinctness (CD) of the image obtained, determined by the relation of each complex trajectory to the linear trajectory of the same angular opening. SC means separating capacity (thickness of cut). It varies with the angle of cut. The exposure time is proportional to the trajectory.

† In linear trajectory, all angles of cut from 10° to 60° can be obtained.

ANTEROPOSTERIOR PROJECTION

With the patient supine and the median plane of the head vertical to the table, a line from the lateral angle of the eye to the tragus also should be vertical to the roentgen-ray table (Fig. 1). The area to be visualized extends approximately from the root of the tragus 15 mm. to the posterior aspect.

Anteroposterior laminagraphy of the normal temporal bone (Fig. 2–6) will reveal in the most ventral cut, taken on a level with the tragus, the external auditory meatus (1) as the most characteristic structure. This may be used as an anatomic point of reference for the initial, cursory study of the laminagram. It is delimited inferiorly by a solid bony shadow representing the tympanic part of the temporal bone, and superiorly by the upper posterior area of the osseous part of the auditory meatus, the so-called "spur" (2) with the tympanic groove. The spur ends anteriorly in the attic as a prominent, broad-based wedge. In cases of lesions in the attic, the spur assumes a rounded shape or an irregular, eroded surface. Thus, this detail of the laminagram should always be inspected very closely.

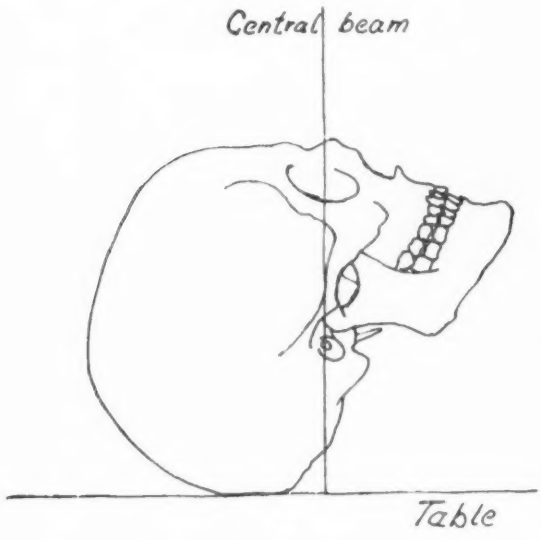

FIG. 1. Diagram showing the direction of the central beam in anteroposterior laminagraphy. (Reproduced with permission from the *Archives of Otolaryngology*.)

The medial part of the external auditory meatus (1) is delimited by the tympanic membrane, normally manifesting itself as a faintly streaked contour. It continues superiorly in the handle of the malleus. The head of the malleus (m) rises in the middle of the attic as a club-shaped shadow. The tympanic cavity (3) presents on the most ventral laminagraphic cuts as a sharply outlined profile, affording good visualization of the hypotympanic recess and promontory, with the spiral-shaped cochlea (4) shown medially. At the site of the upper part of the promontory, just above the cochlea, a portion of the facial

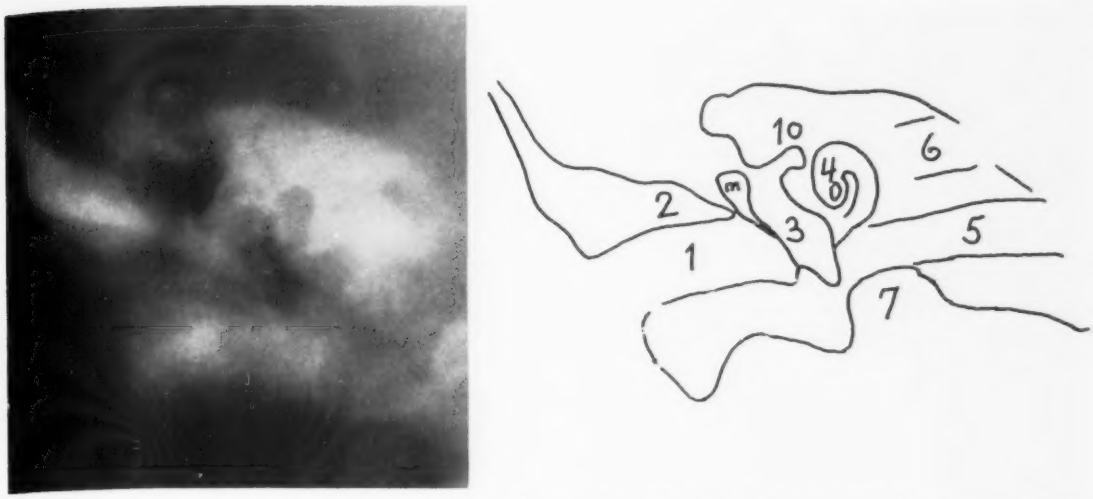

Fig. 2. Anteroposterior laminagram and diagram of a cut through the anterior part of the tympanic cavity (3), showing the malleus (m), spur (2), cochlea (4), external (1) and internal (6) auditory meatuses, pharyngo-tympanic tube (5), jugular fossa (7) and part of the canal for the facial nerve (10). (Reproduced with permission from the *Archives of Otolaryngology*.)

canal (10) is visible, *viz.*, its course from the opening for the facial nerve at the bottom of the internal auditory meatus (6) to the promontory.

The tympanic cavity continues in the attic sharply outlined laterally against the spur and showing on its medial aspect the prominence of the canal for the facial nerve and the lateral semicircular canal (8). Superiorly, the roof is made up of the tegmen tympani, and, since this is only faintly visible or entirely absent on the laminagrams, this lack of visualization need not be a sign of a pathologic condition.

Medial to the cochlea is the internal auditory meatus (6) which appears broad and distinct on the most anterior cuts. In the lateral part of the internal auditory meatus, a small bony crest, the transverse crest, is sometimes visible. Below the in-

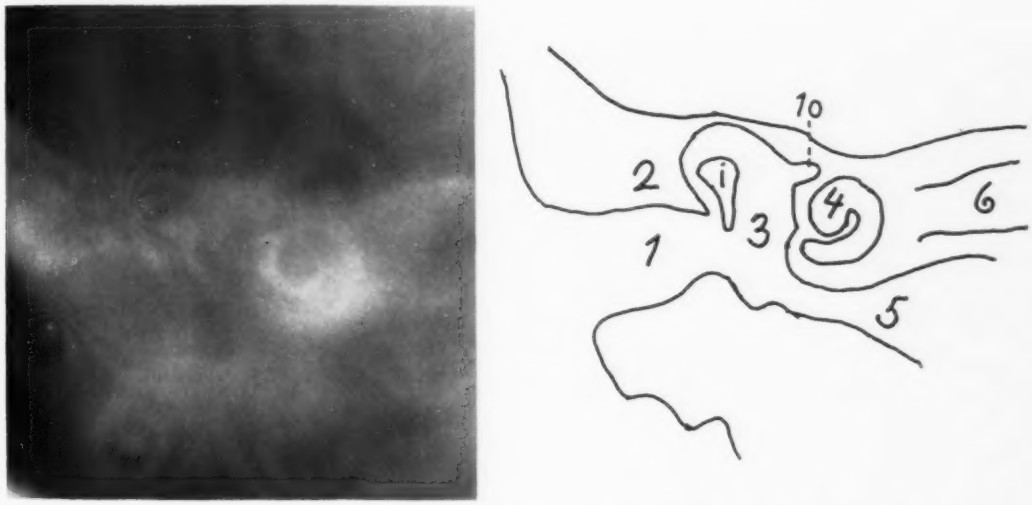

Fig. 3. Anteroposterior laminagram and diagram of a cut made more posteriorly in the tympanic cavity (3) than Figure 2 in which, in addition to the structures shown in Figure 2, the incus (i) may be seen. (Reproduced with permission from the *Archives of Otolaryngology*.)

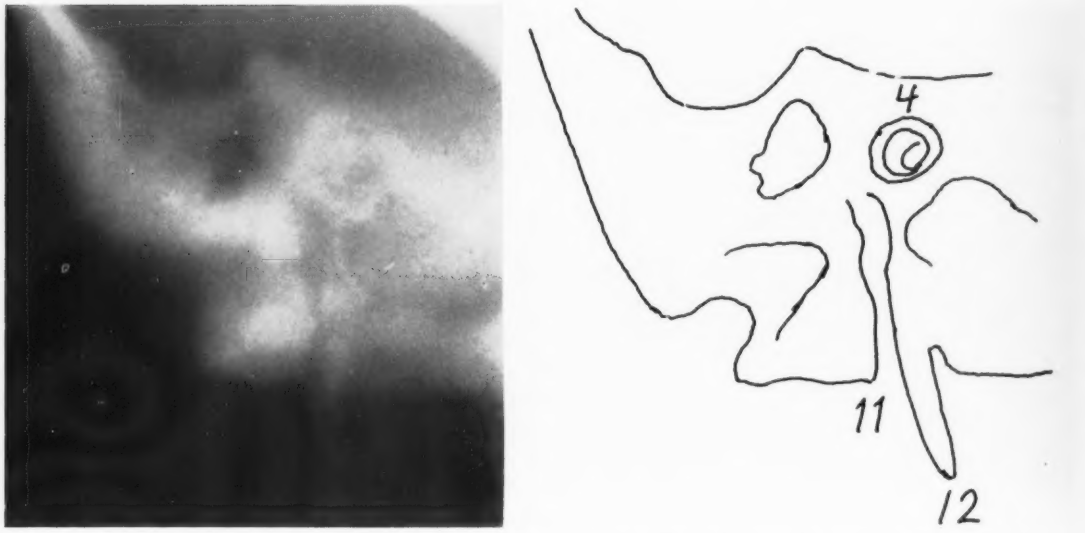

Fig. 4. Anteroposterior laminagram and diagram of a cut through the vertical part of the canal for the facial nerve (11), also showing the styloid process (12). (Reproduced with permission from the *Archives of Otolaryngology*.)

ternal auditory meatus, the pharyngotympanic tube (5) extends from the floor of the tympanic cavity.

On the inferior surface of the pyramid, a single structure may be seen just beneath and medial to the hypotympanic recess, *viz.*, the jugular fossa (7) accommodating the bulb of the jugular vein.

On sections a few millimeters posterior to the tragus, all these structures are generally visible, but the tympanic membrane and malleus (m) have disappeared. Instead, the sections visualize the incus (i) shaped as a short club. Unlike the malleus, the incus is in an extreme lateral position in the attic, its lateral contour extending parallel to the spur. The situation of the two ossicles is extremely important, partly for identification purposes and partly for diagnosing dislocations and congenital deformities. Somewhat more posteriorly, the vertical course of the facial canal (11) may

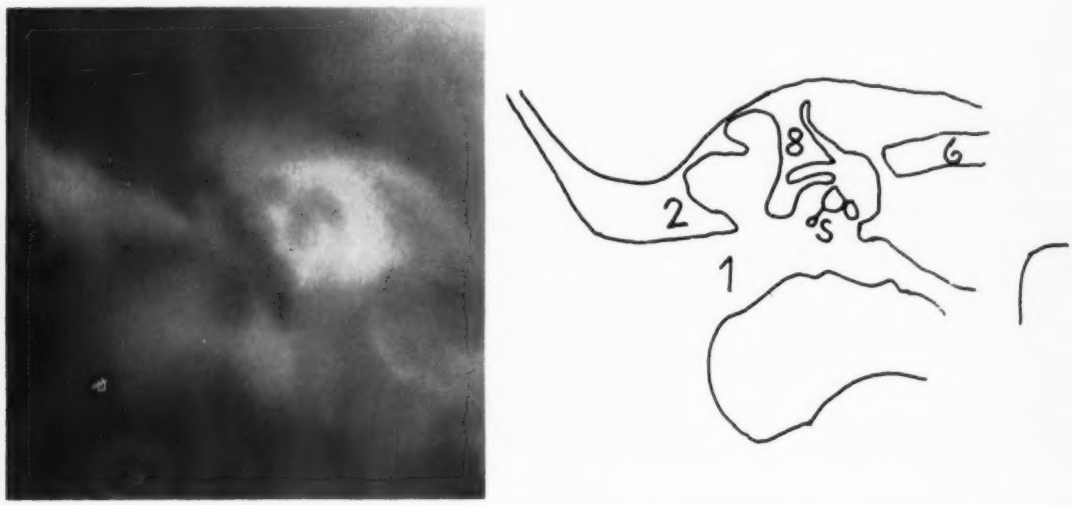

Fig. 5. Anteroposterior laminagram and diagram of a section in the posterior part of the tympanic cavity showing the stapes (s), vestibule, and semicircular canals (8). (Reproduced with permission from the *Archives of Otolaryngology*.)

Fig. 6. Anteroposterior laminagram and diagram of a cut through the mastoid antrum (9) and semicircular canals (8). (Reproduced with permission from the *Archives of Otolaryngology*.)

be seen extending down from the bottom of the tympanic cavity, with its outlet on the inferior surface of the pyramid in the stylomastoid foramen (12).

Cuts just behind the cochlea show the vestibule and semicircular canals (8) most distinctly. The same cut shows the smallest "long bone" of the body, the stapes (s), in part or in full, with the footplate in the oval window which is delimited inferiorly by the subiculum, which, in turn, forms the upper limit of the round window. As yet, we have not found changes indicating ankylosis of the stapes.

On these last-mentioned cuts there is still visible a distinct outline of the attic, or rather the aditus of the antrum, whereas the external as well as internal auditory meatuses have decreased both in size and sharpness, because their posterior bony limit is being reached.

Cuts on a level with the mastoid process (9) still show part of the vestibule and semicircular canals and, at times, a small portion of the internal auditory meatus, although now the external auditory meatus has completely disappeared from view, being replaced by the pneumatic cells of the mastoid process and, superiorly, by the mastoid antrum.

LATERAL PROJECTION

In this projection, the patient is prone with the head turned so that the ear to be examined is up and with both external auditory meatuses in a line perpendicular to the table (Fig. 7). In order to make the patient more comfortable, the shoulder on the side to be examined is elevated, with the homolateral arm flexed and the contralateral arm extended.

Cuts are obtained from the plane of the tympanic membrane, *viz.*, about 2 to 2.5 cm. below the level of the mastoid process and 1 cm. medial, as a rule at a distance of 1 mm. The first cuts visualize the ossi-

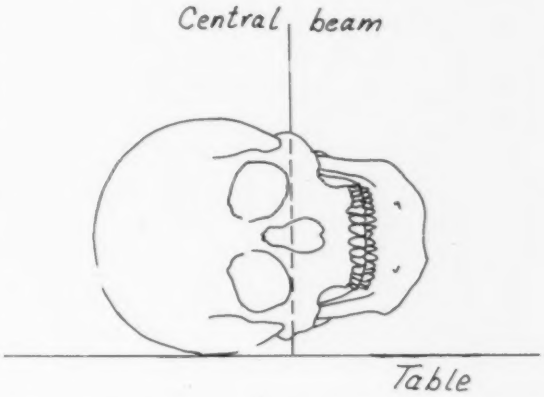

Fig. 7. Diagram showing the direction of the central beam in lateral laminagraphy.

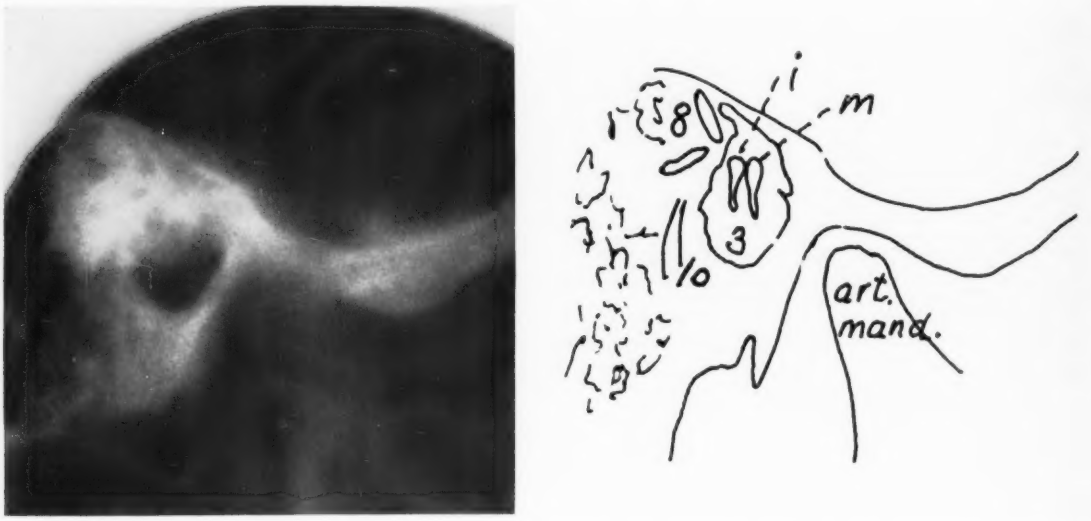

FIG. 8. Lateral laminagram and diagram of a cut through the external part of the tympanic cavity (3), showing the malleus (m) and incus (i) as well as part of the semicircular canal (8) and facial canal (10).

cles (Fig. 8 and 9), the bodies of which are in the attic. The malleus (m) is the most anterior one, its handle projecting down into the tympanic cavity. The body of the incus (i) is immediately posterior to the head of the malleus. The handle of the malleus and the long process of the incus are parallel. Sometimes, the last link of the chain, the stapes may be discerned against the oval window. Superiorly to this, part of the semicircular canal (8) is visualized. Between the latter structure and the tympanic cavity, the upper one-third of the facial canal (10) is often visible. Cuts made more medially will show the distal part of this canal (12).

ILLUSTRATIVE CASES

To demonstrate the diagnostic value of the roentgenographic techniques described

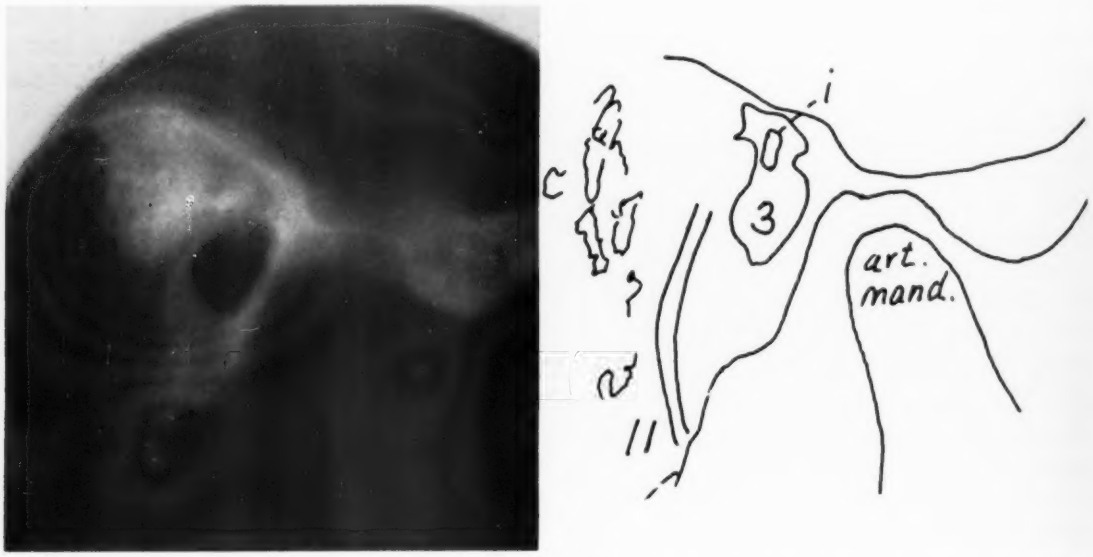

FIG. 9. Lateral laminagram and diagram of a cut through the medial part of the tympanic cavity (3) visualizing the vertical part of the facial canal with the styloid foramen (11).

above, 2 characteristic cases are reported in which laminagraphy was of primary diagnostic importance.

Case 1. This woman, aged seventy years, had been suffering since childhood from chronic, bilateral otitis media with periodic suppuration from both sides when she had colds and sinusitis. Her hearing had been evenly deteriorating, so that for some years she had been using a hearing aid on the left. For the past five years she had had episodes of gyratory and nautic vertigo as well as left-sided tinnitus.

About five months prior to admission, she had experienced a period of severe fatigue, during which total left-sided facial palsy developed within the course of a few days. The condition was interpreted as the sequel to cerebral hemorrhage, and the patient was treated only by bed rest at home. She was admitted for cholelithiasis but, because of the facial palsy, the following investigations were performed.

Neurologic Examination. Results showed left-sided peripheral facial palsy and no sense of taste on the anterior two-thirds of the tongue, the left pupil larger than the right and inactive to light, and diffuse weakening of the limbs with generalized weakening of all reflexes. Romberg's test showed a tendency to fall backward. Cerebrospinal fluid examination was normal. It was difficult to interpret the findings, but an arteriosclerotic lesion in the brain stem was believed probably to be responsible.

Ophthalmologic Examination. This revealed sclerotic changes of the fundus and left-sided hypesthesia of the cornea, presumably due to lagophthalmos. The previously diagnosed ophthalmoplegia interna could not be re-demonstrated, so it must have been transient or drug-induced.

Otologic Examination. There were large, apparently central defects of both tympanic membranes but no discharge or sign of cholesteatoma. The nasolacrimal reflex was present and equal on both sides. An audiogram showed bilateral hearing loss of the conduction type, and nerve deafness consistent with her age. The stapedial reflex could not be tested because of the bilateral chronic otitis.

Roentgen Examination. Using Schüller's, Towne's (slit exposure), and Stenvers' projections, reduced pneumatization with sclerosis of the cells on both sides was revealed but there were no signs of cavitation. Laminagraphy disclosed cavitation on the left side at the site of the anterior portion of the tympanic cavity and the attic, with destruction of the superolateral corner of the cochlea in the horizontal course of the facial canal as well as dilatation of the lateral portion of the internal auditory meatus (Fig. 10 and 11).

Operative Findings. On the basis of the roentgen findings, the patient was subjected to resection of the mastoid process and tympanic cavity on the left with decompression of the left facial nerve. There was pronounced osteitis of the incus, both superficial and profound, and a cholesteatoma in the anterior part of the antrum extending down into the upper portion of the tympanic cavity. At the side of the spur, the facial canal was surrounded by osteitic bone with ample granulation tissue on the sheath.

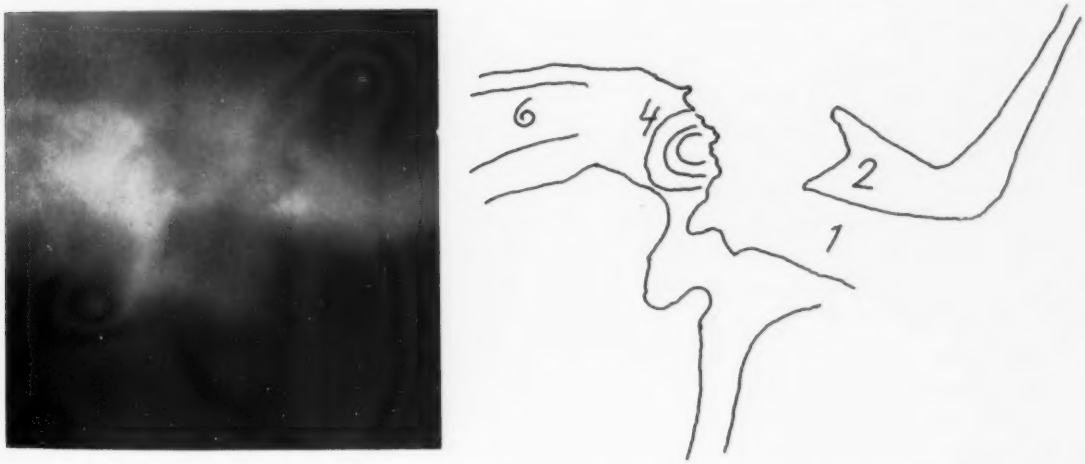

Fig. 10. Case 1. Anteroposterior laminagram and diagram showing destruction at the site of the second part of the facial canal; external auditory meatus (1), spur (2), cochlea (4), internal auditory meatus (6).

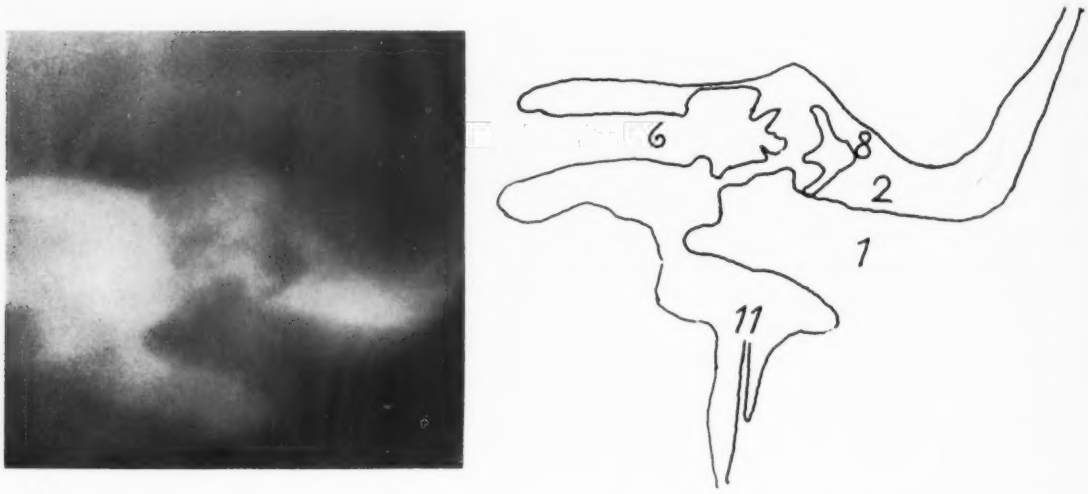

FIG. 11. Case 1. Anteroposterior laminagram and diagram, made more posteriorly than Figure 10, showing that the destruction extends into the lateral part of the internal auditory meatus in the area of the facial nerve; external auditory meatus (1), spur (2), internal auditory meatus (6), semicircular canal (8), facial canal with the styloid foramen (11).

The nerve was hyperemic and showed violent swelling when exposed. It was exposed upwards as far as the geniculate ganglion and downwards as far as the stylomastoid foramen, but no abnormalities were found in these sites.

COMMENT. In recent years, developments in the field of otology, including the increased number of operations performed to improve hearing and the correlative increase in interventions on the facial canal, are making ever greater demands on diagnostic roentgenology.

Routine studies of the temporal bone in the various projections (such as Schüller's, Chaussé's, Stenvers', Runström's, and Towne's—as well as Law's projection based upon those of Stenvers and Schüller) illustrate the pneumatization and disclose the presence of acute or chronic inflammatory changes. In the diagnosis of cholesteatoma or of changes in the ossicles or the facial canal, ordinary roentgen investigation often fails. In 1936, MacMillan[5] reported destructive changes of the temporal bone in only 45 per cent of 288 confirmed cholesteatomas when studying the patients in Law's projection, and in 60 per cent when using Towne's projection. Chiat and Kittredge[3] found destructive changes in 50 per cent also in Towne's projection. In 1960, Brünner, Jensen and Jespersen,[2] in 80 verified cases of cholesteatoma, correctly diagnosed only 46 per cent using Schüller's and the Runström III projections. By employing Chaussé's III projection, the correct diagnoses could have been increased to 70 per cent since this projection increases the possibility of visualizing the antrum and attic, distinct from the labyrinth, and also shows the lateral wall of the attic and external auditory meatus.

By supplementing the usual procedures with laminagraphy, the possibility of demonstrating cholesteatoma could be increased to 80 per cent. This conclusion is supported by a report of Tarp,[8] who demonstrated cholesteatoma by laminagraphy in 19 of 22 verified cases. Thus, laminagraphic study is of great importance, as it affords an opportunity for further advances, especially in visualizing the antrum and/or attic as well as the superior aspect of the external auditory wall and the lateral wall of the attic. In other words, laminagraphy permits a much more exact location of the destruction as well as the damage wrought by it.

There are three important, characteristic findings in the laminagraphic demonstration of changes caused by cholesteatoma: (1) enlargement of the attic and/or antrum and simultaneous destructive changes of the medial or lateral wall of the attic. It has been emphasized by Tarp[8] that enlargement of the antrum to more than 10×12 mm. should arouse suspicion of a cholesteatoma cavity. In our experience, the extent of this enlargement is often very difficult to assess. In order to facilitate the evaluation we introduced the practice, one year ago, of examining both temporal bones by laminagraphy in the anteroposterior projection on the same film. This has supplied us with an extremely important diagnostic aid in evaluating the size of the attic and/or antrum; (2) destruction of the upper medial portion of the external auditory meatus (the spur); in most cases of cholesteatoma involving the attic, it undergoes destruction, while in the presence of changes in the more posterior part of the antral region the spur is often intact; all cases of destroyed spur ought to arouse a suspicion of cholesteatoma and/or osteitis; and (3) destructive change or fragmentation of the ossicles, due in most cases to compromised blood supply, especially to the incus.

Laminagraphy usually visualizes the facial nerve in its entire course through the temporal bone, thus being of valuable supplementary aid in the topical diagnosis of facial nerve lesions. In Case 1, there were destructive changes of the medial wall of the attic as well as around or above the second segment of the facial nerve.

As already mentioned, the projections in current use (Stenvers', Runström's, and Towne's) did not reveal changes other than reduced pneumatization and sclerosis of the cellular systems on both sides, but with no signs of cavitation. Laminagraphy was required to demonstrate the presence of destructive cavitation, especially at the site corresponding to the course of the facial nerve. Surgery could be instituted after the lesion had been fairly accurately located.

Case II. This twenty-three year old man, formerly a sailor, had had a bilateral mastoidectomy at the age of two. Since then, his hearing had been impaired, but he had never had aural discharge, attacks of fever, pain, or recurrent otitis.

Clinical Examination. His general condition

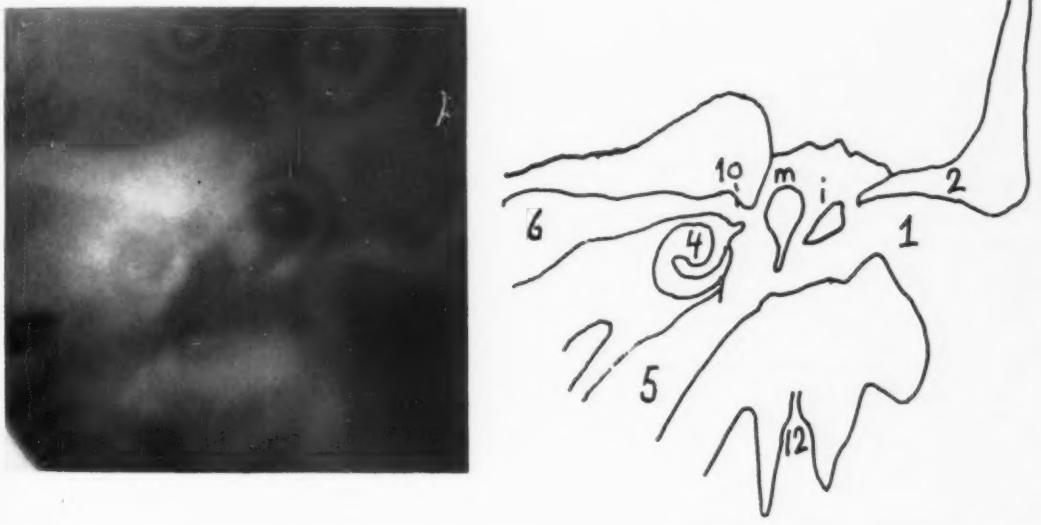

Fig. 12. Case II. Anteroposterior laminagram and diagram of a cut through the anterior part of the tympanic cavity; external auditory meatus (1), spur (2), incus (i), malleus (m), cochlea (4), pharyngotympanic tube (5), internal auditory meatus (6), jugular fossa (7), facial canal (10), styloid process (12).

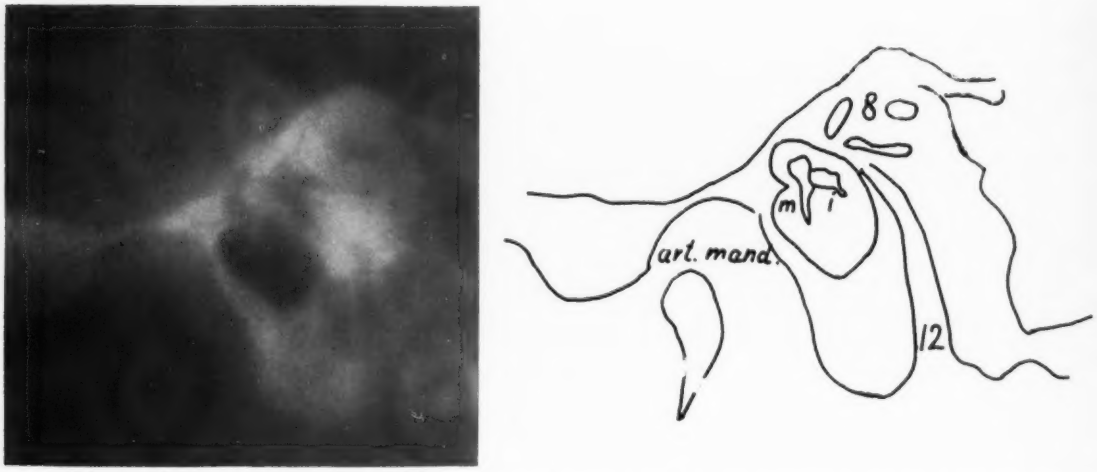

FIG. 13. Case II. Lateral laminagram and diagram of a cut through the tympanic cavity, showing the dislocation of the incus (i); malleus (m), semicircular canal (8), styloid process (12).

was good, although mentally he was severely disturbed by this handicap, and his speech was indistinct and monotonous.

Otologic Examination. The tympanic membranes were grey and lustreless, but intact; no discharge was present in the auditory meatuses. There was hearing of the whispered voice at 0.1 m. and of the spoken voice at 0.5 m. in both ears.

Roentgen Examination. The temporal bones, in the standard projections used in Case I, showed the sequelae of mastoidectomy on both sides with no signs of destructive changes. Anteroposterior and lateral laminagraphy (Fig. 12 and 13) showed on the left that the body of the incus had slid down, so that the long process of the incus was horizontal. It was impossible to locate the stapes definitely. On the right side, the head of the malleus was distinct, but the body of the incus and its long process were blurred and appeared to be dislocated. On both sides operative cavities left by simple mastoidectomy were seen.

Operative Findings. Surgery was performed on the left ear, on October 17, 1959. After exposing the middle ear, the incus was seen to be displaced, with its long process pointing outward and forward instead of downward and medially. This interrupted the connection between the incus and stapes, there being a distance of 2–3 mm. between the two joint surfaces. It proved possible to reduce the incus and successfully restore the normal communication between the joints.

The postoperative course was uneventful despite the fact that, while incising the auditory meatus, an opening into the antrum could not be avoided, because the posterior wall of the external auditory meatus had been removed at the previous operation. The patient's hearing was restored to practically normal, with the hearing of the whispered voice increasing from 0.10 m. to 4.5 m.

Operation on the right ear was performed on November 13, 1959. After opening the middle ear, it was found that the situation was less favorable than it had been on the left, the body of the incus being turned posteriorly almost 90°. This had completely interrupted the connection between the malleus and stapes, and the incus was firmly impacted between the facial canal and the lateral wall of the attic. Restoring the ossicle chain was out of the question. Instead, the tympanic ring was freed from the tympanic groove in the posterior two-thirds of the circumference, so that the tympanic membrane could be wrapped around the intact and freely mobile stapes.

The hearing of the whispered voice improved from 0.1 m. to 2 m. within three weeks. In other words, the hearing improvement in this ear too was of great practical importance.

COMMENT. Today it is of the utmost importance to be able to diagnose morbid processes in the temporal bone at a stage so early that inoperable changes of the facial nerve and vestibular apparatus may be prevented. If plastic operations for improving the hearing are to give a favorable

result, the processes must not have had time to interrupt the blood supply to the ossicles or to cause major bone destruction, since it is the object of tympanoplastic operations to reconstruct an intact chain of ossicles and restore to the patient a degree of hearing sufficient for practical purposes.

The increase in recent years in the frequency of cranial injuries due to traffic accidents resulting in fractures of the base of the skull has also increased the number of traumatic changes in the ossicles and their conduction links. As early as 1955, Welin[9] stressed the great importance of laminagraphy of the ossicles in cases of infection and especially in traumatic injuries. It is beyond doubt that, in numerous cases of cranial injury, more attention has been devoted to the immediate vital problems of a more general nature at the cost of the otologic changes which have led to hearing impairment.

Hearing impairment following operations performed a number of years ago for otitis was probably due in many cases to the less advanced operative and anesthetic techniques of those times. This is illustrated by our Case II. In childhood the patient had undergone simple bilateral mastoidectomy after which his hearing had been greatly impaired. Laminagraphic investigation revealed displacement of the ossicles, with dislocation in the incostapedial joint. Bilateral, exploratory tympanectomy revealed, in accordance with the roentgen findings, dislocation of the incus on both sides. After reduction of the left incus, an anatomically correct position of the ossicles was obtained. On the right, the incus could not be reduced, as it was firmly impacted. Instead, myringostapediopexy was performed. The operative treatment improved the hearing in the right ear considerably, but also to some extent in the right ear, so that almost normal hearing was restored.

Laminagraphy of the ossicles will no doubt gain ever increasing practical importance, not least because of the increased number of traffic accidents. It will contribute to diagnosing displacement of the ossicles caused by the more primitive techniques formerly used in aural surgery.

SUMMARY

A laminagraph with polycyclic movements and excursions wide enough to permit thin and well-defined cuts is discussed. By means of this apparatus, anteroposterior as well as lateral views of the temporal bones were obtained. A description of the normal sections and the characteristic pathologic findings is given.

In connection with a case of cholesteatoma, the most characteristic findings are stressed: (1) destruction of the spur; (2) dilatation of the antrum and/or attic demonstrated by comparison with the normal side; and (3) destruction or fragmentation of the ossicles.

A case of dislocation of the ossicles illustrates the importance of lateral laminagraphy in diagnosing changes in the position of the ossicles.

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CYSTS AND TUMORS OF THE MANDIBLE*

THEIR DIFFERENTIAL DIAGNOSIS

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RADIOLOGISTS have over the years taken an increasing interest in roentgenography of the mandible, devoting their attention to systemic diseases and malignant tumors. While dentists and dental surgeons are primarily interested in odontogenesis, it is recognized that there is an overlapping of interests, and hence the roentgenographic appearances of odontogenic defects, mandibular diseases, and tumors should be equally familiar to both radiologists and their dental colleagues.

The mandible participates in three essential functions, *i.e.*, mastication, deglutition, and odontogenesis. Since odontogenesis is peculiar to the mandible, the pathologic changes occurring during this process must be recognized and carefully considered in the differential diagnosis.

Many classifications of mandibular cysts and tumors have been suggested. The difficulty in selecting an appropriate one stems from the complex histologic appearance of the lesions and the uncertainty of tissue of origin. In some tumors, such as ameloblastomas, there is no unanimity of opinion as to the benign or malignant potentiality of the entity. It is not the authors' intent to introduce another classification, but instead, Table 1 is to be used to provide a systematic approach to the lesions that are more frequently encountered.

In this paper the common cysts and tumors affecting the mandible are reviewed and those characteristics that may aid in differential diagnosis are discussed.

EMBRYOLOGY

In the sixth fetal week epithelial or ectodermal cells in the embryonic area of the mandible begin to multiply and extend downward into the adjacent mesoderm (Fig. 1, *1*). The mesoderm then responds by forming collections of cells called dental papillae which grow up into the descending ectodermal cells (Fig. 1, *2*). As the two meet, the ectodermal cells form a caplike cover over the dental papillae—the ectodermal cells will form the enamel while the mesodermal dental papilla will form the dentin and pulp of the tooth. Each embryonic tooth is cut off from any connection to the surface epithelium by a membrane of vascular connective tissue (periodontal membrane) which grows upward and completely encloses the dental germ (Fig. 1, *3*). The periodontal membrane eventually becomes a specialized periosteum, lining the tooth socket and connecting the tooth to the mandible. The mandible proper is formed from the paired Meckel's cartilages, which meet anteriorly in the midline, forming the mandibular symphysis. As the tooth bud matures the ectodermal cells divide into three layers. Those immediately adjacent to the mesodermal papilla are the enamel cells or ameloblasts. The outer layer forms the external enamel epithelium while the middle layer differentiates into a stellate network and secretes a fluid enamel pulp (Fig. 1, *4–6*). It is at this stage of differentiation that anomalies of development commonly occur.

DISCUSSION

1. MANDIBULAR CYSTS

Most mandibular cysts, except for the rather controversial type that has been

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called "traumatic," are epithelium-lined. These are thought to increase in size because of the tension of the fluid or semisolid contents;[15] thus, they are usually globular due to the equal pressure in all directions. The cysts may be either single or multiple and may coalesce or remain separate. Some contain calcific material which will often give a clue as to the roentgenologic diagnosis. They consist of four types.

1. *Periodontal or Radicular Cysts.* Infections of the tooth pulp often spread through the pulp canal into the adjacent mandible,[15] forming a dental granuloma which appears roentgenographically as an oval radiolucent area at the tip of the pulp canal. This inflammatory response causes proliferation of epithelial remnants in the periodontal membrane, with subsequent formation of epithelium-lined cysts (Fig. 2). On roentgenograms, it is extremely difficult to differentiate between a granuloma and these radicular cysts even though the latter are supposedly more radiolucent with a well circumscribed, often slightly sclerotic, margin.[7] Stafne[15] does not believe that granulomas and radicular cysts of equal size can

TABLE I

CYSTS AND TUMORS THAT AFFECT THE MANDIBLE

I. Cysts
   1. Periodontal or Radicular
   2. Squamous
   3. Dentigerous
   4. Traumatic
II. Tumors
   A. Odontogenic Tumors
      1. Ameloblastoma or Adamantinoma
      2. Odontoma
      3. Cementoma
      4. Enameloma
      5. Myxoma
      6. Fibroma
   B. Nonodontogenic Tumors
      1. Osteoma
      2. Osteoid Osteoma
      3. Chondroma
      4. Neurofibroma
      5. Hemangioma
   C. Tumors of Indeterminate Origin
      1. Giant Cell
      2. Fibrous Dysplasia
      3. Reticuloendotheliosis
      4. Infantile Cortical Hyperostosis
   D. Malignant Tumors
      1. Primary Malignant Tumors
      2. Metastases
      3. Extension from Surrounding Tissues

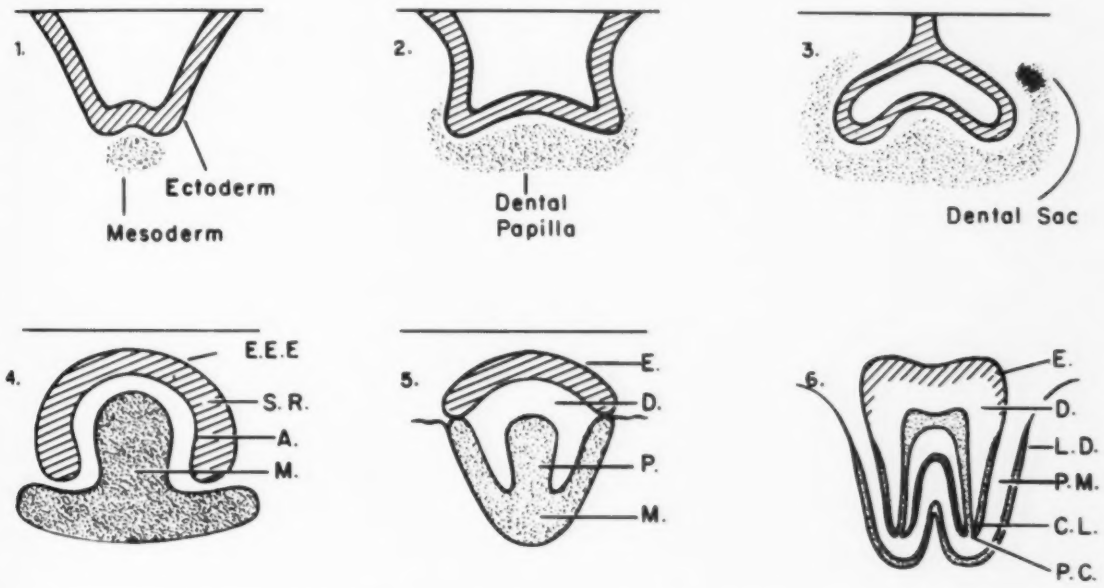

FIG. 1. Diagrams *1–6* illustrating embryologic development of a tooth. E.E.E.—external enamel epithelium; S.R.—stellate reticulum; A.—ameloblasta; M.—mesoderm; E.—enamel; D.—dentin; P.—pulp; L.D.—lamina dura; C.L.—cementoblastic layer; P.C.—pulp canal.

Fig. 2. A twenty-nine year old man had some aching in the right mandible. Following roentgenographic examination the cystic area was surgically opened and thick purulent material was expressed. The cavity was epithelium lined. The pathologic diagnosis was radicular cyst.

be differentiated by roentgen examination. The radicular cysts represent a common lesion, with the highest incidence in the third and fourth decades of life. More men than women are affected.

2. *Squamous Cysts.* In this category are placed the cysts that have no calcific deposits within them and are found at biopsy to have a squamous or epithelial lining. Any ectodermal tissue may give rise to squamous cysts. Thoma[17] suggests that these odontogenic cysts are formed from remnants of oral epithelium, proliferation of the dental lamina or from the enamel organ of a developing tooth. Jaffe[7] believes that these cysts are not associated with infection or maldevelopment of the dental germ but are formed when epithelial cells become trapped in the unfused mandible. The squamous cysts may be either monolocular or multilocular (Fig. 3 and 4). Usually, they have a relatively well-defined margin. In some cases the multilocular appearance is due to the lobulated margins of a single cavity (Fig. 5, *A* and *B*).

A rare type of squamous cyst, thought to have a different pathogenesis, is the primordial cyst that develops from the dental sac before any of the ectodermal or mesodermal cells become sufficiently differentiated to produce calcification. It is practically impossible to distinguish it from a common squamous cyst, being usually monocystic with a thin sclerotic margin. Primordial cysts are principally found in the second to sixth decades and affect both sexes equally. Thoma[17] believes that they are usually more common near the angle of the mandible where they sometimes extend into the ramus.

3. *Dentigerous Cysts.* Since these cysts develop from the enamel organ during or shortly after formation of the crown, they are associated with an unerupted tooth. Some authors[10,15] believe that these cysts are caused by an accumulation of fluid

Fig. 3. A sixty-three year old woman with mandibular swelling for two years and local pain for one year. The pathologic diagnosis was squamous cyst. Note the unilocular appearance.

Fig. 4. A forty-four year old man with a two year history of aching in the left temporomandibular region when in a draft. The pathologic diagnosis was squamous cyst. Note the multilocular appearance.

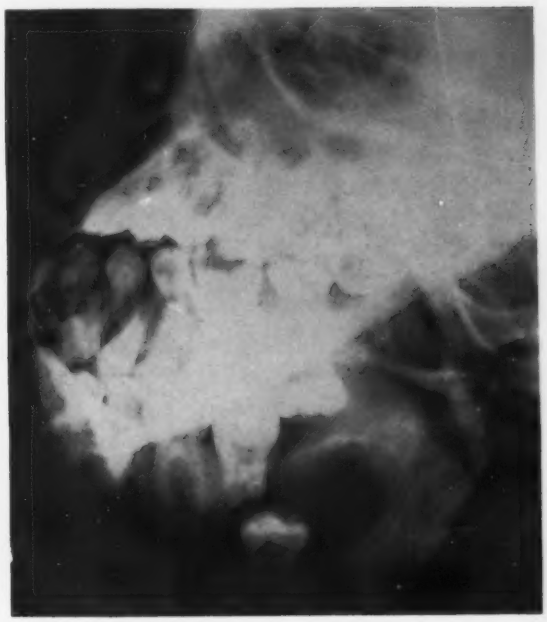

Fig. 6. An eighteen year old, asymptomatic female was found to have a cyst on routine dental roentgenograms. At operation the diagnosis of a dentigerous cyst was confirmed. Note the orientation of the cyst to the crown of the tooth.

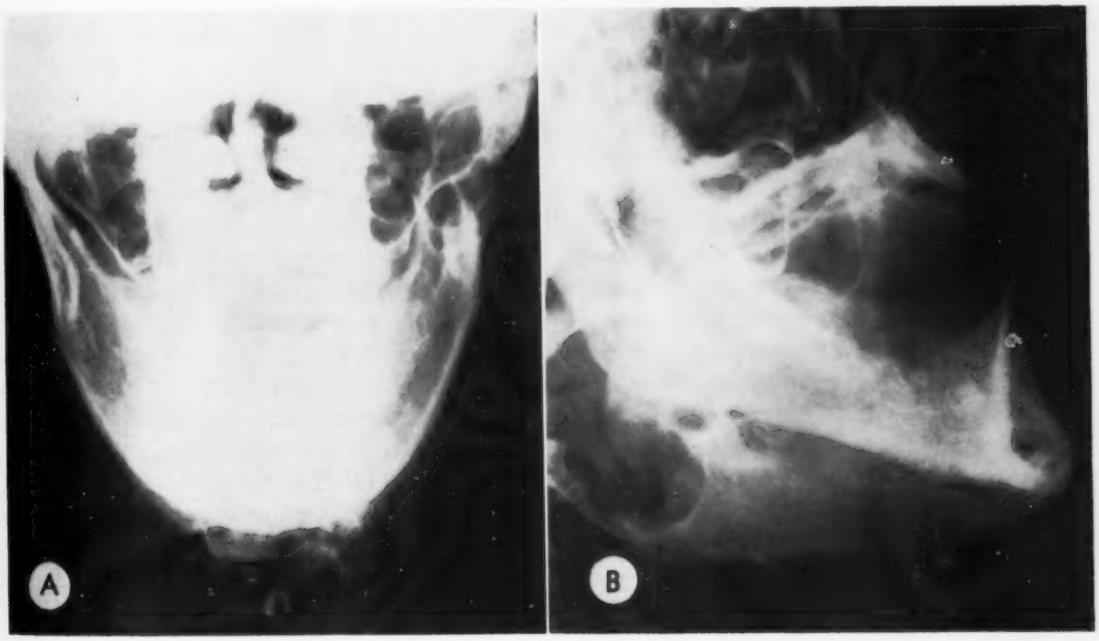

Fig. 5. A sixty-seven year old Negro woman with a fourteen year history of a left mandibular swelling. The pathologic diagnosis was squamous cyst. (*A*) Note the multilocular appearance on the postero-anterior roentgenogram. (*B*) Oblique roentgenogram showing the true single cavity.

between the enamel and the surrounding soft tissue capsule while others[7] think that the fluid within the enamel anlagen is responsible. In either event the onset is early in life, usually in the first two decades, and Stafne[15] is inclined to think that there is a hereditary tendency. The cysts are predominantly single. The roentgenographic appearance is that of a monolocular cystic lesion enveloping the crown of a normal appearing tooth (Fig. 6). Usually there is a clearly defined margin, and those in the molar region are more likely to have a sclerotic rim.[14]

4. *"Traumatic" or Nonepithelium-lined Cysts.* These cysts are lined with connective tissue instead of epithelium. One opinion[15] is that trauma may be the etiologic factor causing an intramedullary hematoma which as it degenerates leaves a cystic area. There is some doubt that trauma is always the etiology, and it has been suggested that these cysts may result from an aberration in growth and development of the local osseous tissue.[7] The borders of these cysts are not well defined and are more irregular than those of epithelium-lined cysts. The incidence is highest in patients between the ages of ten and twenty years, and the location of the cyst is usually in the posterior body of the mandible.

II. MANDIBULAR TUMORS

Tumors of the mandible may be divided into four categories: odontogenic, nonodontogenic, those of indeterminate origin and malignant. The odontogenic type includes those that arise from dental germ, while the nonodontogenic group consists of tumors of the bone, cartilage, nerve and vascular constituents of the mandible that are not involved in odontogenesis. The indeterminate origin group contains lesions of speculative etiology, and those closely simulating mandibular tumors in appearance. The malignant tumor category encompasses the primary malignant tumors, metastases and tumors involving the mandible by direct extension from the surrounding soft tissues.

In general, the roentgenographic appearance of benign and malignant tumors of the mandible is similar to that of these tumors when they occur in other bones in the body. The margins of benign tumors are usually well defined;[15] these tumors have an intact cortex even though it may be thinned and expanded; and they more frequently cause resorption of tooth roots, possibly attributable to their comparatively slow growth rate. Malignant tumors, of course, are the antithesis of the benign tumors, and, in addition, may have a periosteal reaction or "sunburst" appearance.

A. ODONTOGENIC TUMORS

These tumors arise from the cells involved in odontogenesis. On microscopic examination, their cytologic pattern suggests an origin from mesoderm, ectoderm or both. Some have a roentgenographic appearance of diagnostic significance, notably the "calcified odontomas," the encapsulation of which in most cases is visualized as a thin radiolucent area at the circumference of the tumor.[15]

1. *Ameloblastoma or Adamantinoma.* Histologically, these tumors show wide variations but in practically all cases cells resembling the stellate reticulum of the enamel organ can be identified.[17] Stafne[15] believes that they arise from ectodermal cells in enamel organ, periodontal membrane, follicle, or in the epithelial lining of dentigerous cysts.

An alternate theory is that ameloblastomas arise from the "debris of Malassez,"[17] or cells that originally were in the tubular enamel structure called Hertwig's sheath. This sheath forms the tooth roots and then disintegrates; any remaining cells will then lie in the periodontal membrane as the "debris of Malassez."[13]

Clinically, they produce painless expansion of the mandible in young adults. The frequency is somewhat higher in the Negro race. The site of origin is frequently in the angle of the mandible with extension into the ramus (Fig. 7).[14]

The roentgenographic appearance is varied. Ameloblastomas may be monolocular

and may resemble any type of cyst. A more typical appearance is that of multiple small radiolucent areas, which are variable in size and simulate a cluster of grapes (Fig. 8, *A*, *B* and *C*). These areas are separated by distinct septa.[16] The cortex usually is only expanded but destruction and invasion of the soft tissues may be present.

Jaffe[7] believes that these tumors are benign but may recur or occasionally metastasize. Robbins,[13] on the other hand, questions whether adamantinomas are malignant even though some metastases have been claimed. He believes that they are locally destructive benign tumors which tend to recur following incomplete surgical treatment.

2. *Odontoma.* Odontomas are considered to contain all the elements of dental structure, such as dentin, enamel, pulp, and cement. They occur in the area in which there has been failure of formation of a normal tooth. Usually, they contain variable amounts of calcification.

An important roentgenographic finding is a radiolucent area of varying width surrounding the tumor. This is due to a fibrous capsule separating the tumor from adjacent structures.

Odontomas as a group are called "composite odontomas,"[13] which are further divided into complex and compound types. The complex composite odontoma is roentgenographically seen as a mass of irregular calcification in a globular arrangement with a distinct separation from the surrounding bone (Fig. 9). There is no resemblance to actual tooth formation. The compound composite odontoma differs from the former in that it contains calcified dental tissue that actually resembles small teeth (Fig. 10), the number being variable. Either type may expand the mandibular cortex.

3. *Cementoma.* Cementomas arise from the periodontal membrane of fully erupted teeth. The membrane proliferates into fibrous connective tissue that calcifies. Because of the hyperplasia of fibrous tissue, the early roentgenographic appearance is

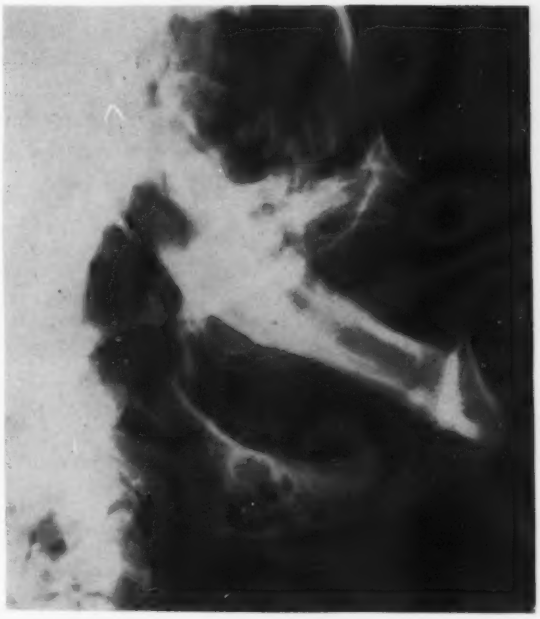

Fig. 7. A thirty-three year old woman with a one year history of sharp pain in the left jaw, radiating to the temple. At operation the pathologic diagnosis was ameloblastoma.

that of a radiolucent cavity which progressively changes to a characteristic calcified mass *attached* to the tooth root and surrounded by a small radiolucent cavity.[10,15] These relatively frequent tumors are not diagnosed often because they are usually asymptomatic. They occur in persons from twenty to sixty years of age and are slightly more common in women and in Negroes.[3]

4. *Enameloma.* These lesions possibly arise from remnants of the enamel anlage cells. They are rare and appear roentgenographically as small opaque tumors at the bifurcation of the tooth roots. They are smaller than odontomas and more radiopaque than cementomas.[3]

5. *Myxoma.* These tumors arise from the mesenchymal portion of the dental germ and histologically contain myxomatous connective tissue.[15] The roentgenographic appearance is varied but may simulate that of dentigerous cysts by being associated with unerupted teeth. In some instances myxomas are found in an area where there is congenital absence of teeth; this supports the contention that these tumors differ

from other myxomas and are odontogenic in origin.[17] Myxomas are partially encapsulated, and when large, give a "honeycombed" appearance which simulates an ameloblastoma.

6. *Fibroma.* Histologically, fibromas contain a few ectodermal cells and occasionally some calcification;[15] however, they arise from the mesenchymal portion of the dental germ. Roentgenographically, they appear as relatively well-defined radiolucent areas in close apposition to the crown or root of

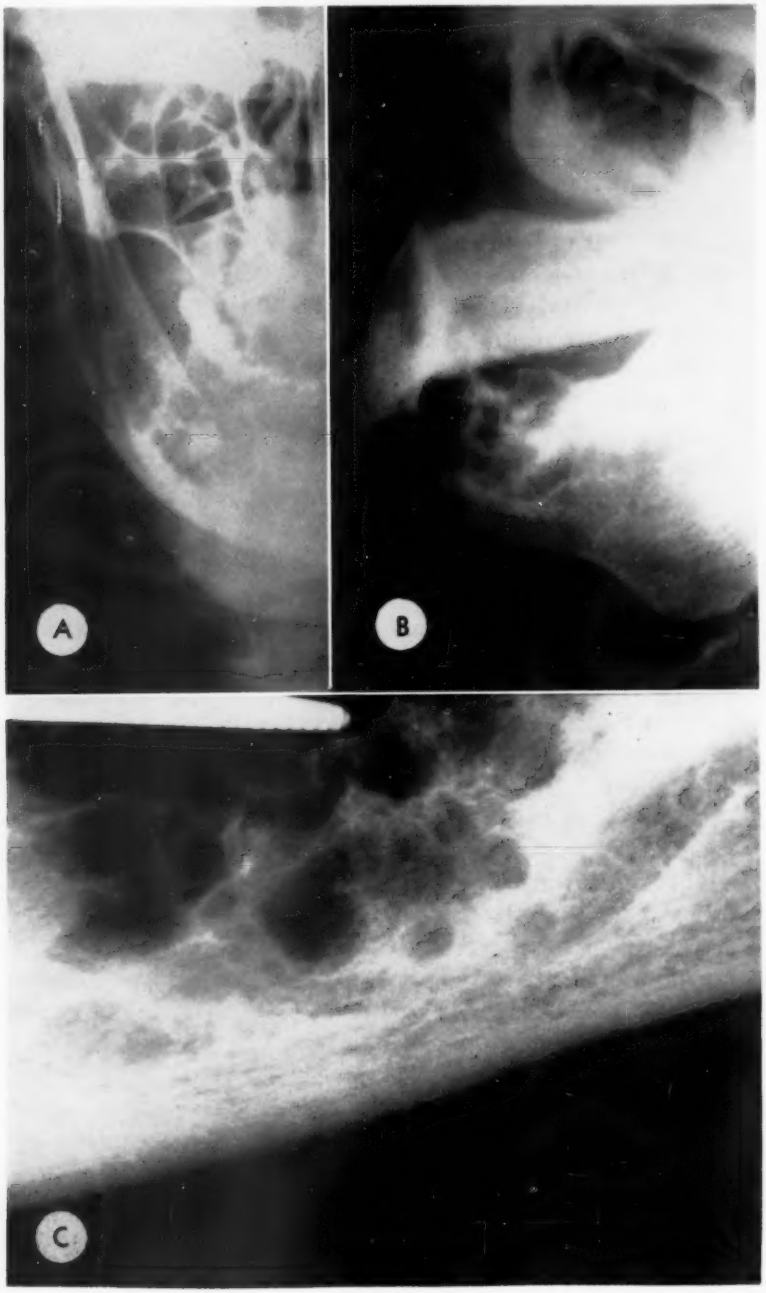

FIG. 8. (*A, B* and *C*) A seventy-two year old woman with a fifteen year history of asymptomatic swelling of the right mandible that gradually increased in size. The pathologic diagnosis from a specimen removed at operation was ameloblastoma.

the tooth, thus simulating radicular or dentigerous cysts. These tumors are classified by some authors as a variant of fibrous dysplasia.

B. NONODONTOGENIC TUMORS

These tumors are histologically similar to tumors of nerve, vascular, bone and cartilaginous tissues elsewhere in the body. Roentgenographically, the criteria for diagnosis are much the same when these tumors involve the mandible.

1. *Osteoma.* This is a rare benign tumor that is seen at the angle of the mandible, originating in the subperiosteal layers without invading the medullary bone.[10] The tumor may be on a pedicle or have a wide base,[15] with a density varying from very opaque to that of normal bone. Trabeculae may be seen to extend from the surrounding bone into the tumor; occasionally, the margins are corticated.[14]

2. *Osteoid Osteoma.* The majority of these tumors are cortical or just subcortical. Local pain is a prominent feature when they occur in the mandible as it is in other bones. The roentgenographic picture is that of a sclerotic nidus in a radiolucent area. The surrounding bone appears more dense than normally.[15]

FIG. 9. A thirty-six year old man with a three week history of swelling of the left side of the face. There was no numbness but he had some local pain. At operation the diagnosis of complex composite odontoma was confirmed. This is an unusually large lesion. Note the clear space surrounding the tumor, indicating encapsulation.

FIG. 10. A twenty-nine year old man in whom a compound composite odontoma was an incidental finding on routine dental survey roentgenograms. The patient had no symptoms referable to the mandible.

3. *Chondroma.* Chondromas are tumors that are primarily cartilage but because of osteoblastic activity may evolve into an osteochondroma or osteoma. The roentgenographic appearance is that of a cortical, raised radiolucent tumor with irregular areas of calcifications. Even though there may be a well-defined sclerotic rim, these tumors should be considered potentially malignant.

4. *Neurofibroma.* These tumors present a complex histopathologic picture. For the radiologist's purpose, they may be considered to be benign tumors that originate from the sheath of a peripheral nerve. In the mandible they characteristically arise in one of two locations. The first is in the mandibular canal, where the tumor will produce an oval expansion or radiolucent area with a fairly well-defined periphery.[15] This tumor is actually a fusiform expansion of the mandibular branch of the fifth cra-

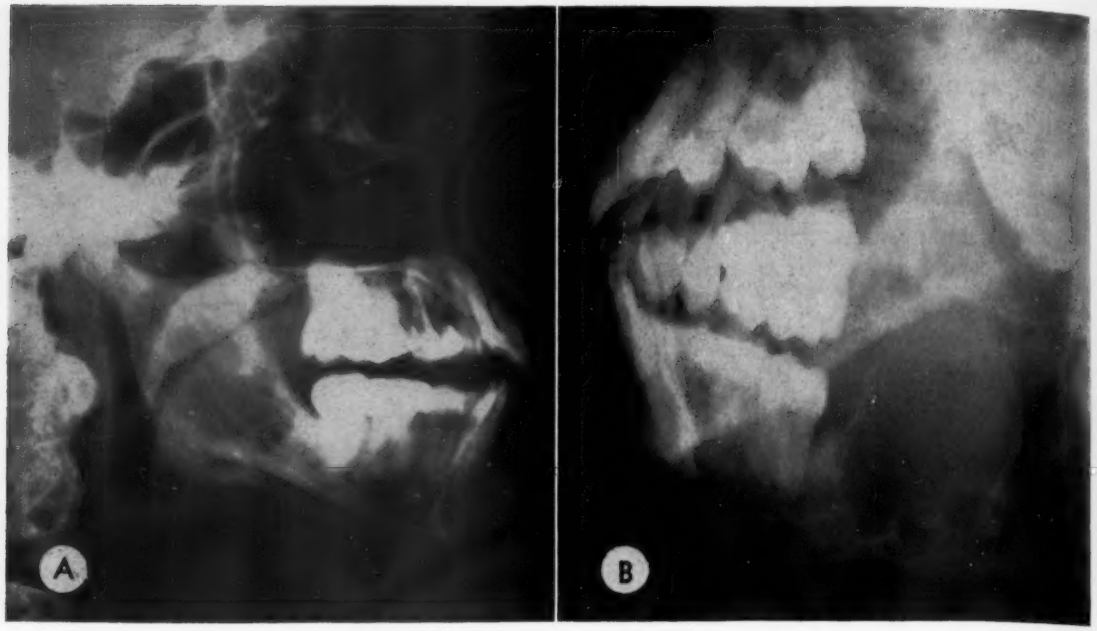

Fig. 11. (*A* and *B*) A twenty year old female with a one year history of a painful swelling of the upper gum. Roentgen examination of the mandible showed multiple osteolytic areas. Several teeth were loose. The serum calcium was 19.3 mg. per 100 ml., the serum phosphorus, 3.1 mg. per 100 ml. Needle biopsy specimens of the mandible, showing numerous giant cells, were consistent histologically with the diagnosis of hyperparathyroidism. A functional parathyroid adenoma was removed surgically.

nial nerve. The second site of development is in one of the periosteal nerves. As the tumor enlarges, it will cause erosion of the adjacent bone with formation of a pitlike area. It may have a thin shell of bone covering the tissue, thus giving it a cystic appearance.[10]

5. *Hemangioma.* Hemangiomas are vascular tumors that contain varied amounts of connective tissue stroma and in bone commonly arise in the spongiosa. The two histologic types are capillary and cavernous, both being rare. Clinically, they may produce bone expansion or pain. Of the few reported in the medical literature, the majority have been in young adults. If, on roentgenograms, coarse trabeculae can be seen to extend at right angles from the surrounding bone into the lesion, Stafne[15] feels that this is pathognomonic of hemangiomas. Pizer[11] reports a case of cavernous hemangioma in which the tumor appeared as an ill-defined radiolucent area without expansion. In another case of cavernous hemangioma,[2] expansion of the cortex with coarse long bony trabeculae was demonstrated suggesting an osteogenic as well as an osteolytic process. This hemangioma showed the more typical bony characteristics which occur when this tumor involves the vertebral bodies.

c. Tumors of indeterminate origin

As the term implies, these entities do not have a clear etiopathogenesis; they may, in fact, not represent true tumors. However their roentgenographic appearance so closely resembles that of tumors that they are included as "tumors of indeterminate origin."

1. *Giant Cell Tumors.* These tumors microscopically are shown to contain multinuclear giant cells, a feature shared with several other tumors. Jaffe[7] believes that most of the previously diagnosed giant cell tumors of the mandible were either the "brown tumor" of hyperparathyroidism (Fig. 11, *A* and *B*) or giant cell reparative granulomas that are a response to inflammation. He feels that the neoplastic giant

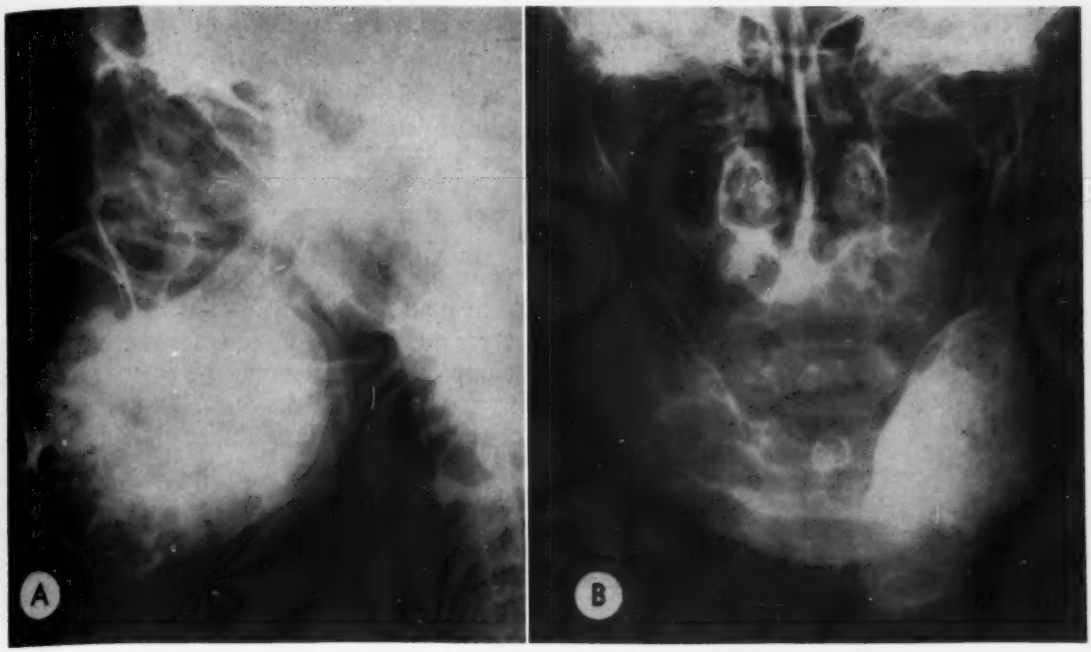

FIG. 12. (*A* and *B*) A fifty-three year old woman with bilateral mandibular swellings for seventeen years. The pathologic diagnosis was osteogenic fibroma.

cell tumor seen in other bones is exceedingly rare in the mandible. Roentgenographically, mandibular giant cell tumors have an extremely varied appearance. They may appear either as a single area of destruction with no septa or as multilocular lesions with septa that are more numerous and not as distinct and regular as those in ameloblastomas.[16] Jaw expansion is common.[14]

According to Jaffe[7] giant cell reparative granulomas occur between the ages of ten and twenty-five years, while giant cell tumors are found in patients over twenty years of age.

2. *Fibrous Dysplasia.* The classification of the fibro-osseous lesions is a difficult task. The term "fibrous dysplasia" has been used by many authors with various meanings. Some classify osteofibromas (Fig. 12, *A* and *B*), ossifying fibromas, and fibro-osseous dysplasia as variants of fibrous dysplasia. Zimmerman, Dahlin, and Stafne[19] describe fibrous dysplasia histologically as a tumor of connective tissue with active osseous metaplasia. They believe that it is a benign fibro-osseous process that involves the spongiosa of the bone causing expansion and deformity. It is a disease of unknown etiopathogenesis, thought to be more common in persons in the second and third decades of life, with approximately 70 per cent of the cases in females.[19] The roentgenographic appearance may be quite varied—from a unilocular well-defined lesion (Fig. 13) (monostotic fibrous dysplasia) to a multilocular mottled and sclerotic lesion (polyostotic fibrous dysplasia). Both the unilocular and the multilocular types often contain poorly defined sclerotic areas that give an over-all appearance of "smudging," which is almost pathognomonic (Fig. 14). It rarely may appear as a very radiopaque tumor with irregular margins and may become almost imperceptibly fused with the bone.

Caffey and Williams[1] believe that familial fibrous swelling of the jaws is a separate entity and should be classified as such. They attribute the term "*familial* fibrous dysplasia" to Dr. W. H. Jones, who coined the words "cherubism" and "eyes raised to heaven" look. Cherubism produces a mas-

Fig. 13. A twelve year old girl with an asymptomatic swelling of the right jaw for eight days. The pathologic diagnosis was fibrous dysplasia. Note the monolocular appearance and the "smudging."

Fig. 14. A forty-eight year old man who had had a partial nasal obstruction at twelve years of age, after which all of the bones of the left side of the forehead and face increased in size. Serum calcium, phosphorus and phosphatase levels were normal. The diagnosis was fibrous dysplasia.

sive bilateral swelling of the cheeks and jaws which stretches the skin, pulling on the lower eyelids and giving the "eyes raised to heaven" look. It begins in the first three years of life.[10] The roentgenographic appearance is that of bilateral multilocular expansion of the mandible with thinning and expansion of the cortex (Fig. 15; and 16, *A*, *B* and *C*). The swelling is firm and painless, and improves markedly after puberty. In some cases there is almost complete restoration of the normal features.

3. *Reticuloendotheliosis.* The term "reticuloendotheliosis" includes the lesions of Letterer-Siwe disease, Hand-Schüller-Christian disease and eosinophilic granuloma. Pugh[12] considers all three to be variations of the same disease but of different severity of involvement. The roentgenographic appearances in the mandible are similar: numerous rather sharply defined but not corticated radiolucent areas. The characteristic picture is that of a sharply outlined destruction of the alveolar ridge with the unusual finding of teeth that are not imbedded in bone but appear to be "floating in air" (Fig. 17, *A* and *B*; and 18).

Fig. 15. An eight year old boy with an asymptomatic bilateral swelling of the jaw for three years. All siblings were normal. The pathologic diagnosis was fibrous dysplasia (cherubism). Note the bilateral cystic involvement.

4. *Infantile Cortical Hyperostosis.* This disease is a self-limiting process of obscure etiology which appears between the ages of three and six months. Clinically, the infants have fever, irritability, leukocytosis and anemia. Roentgenographically, the mandible, ribs, clavicles, and tubular bones show subperiosteal bone lamination with some cortical thickening.[18]

D. MALIGNANT TUMORS

Carcinomas are unusual and probably always arise from coexisting abnormalities. For example, Figure 19 shows a muco-epidermoid cyst that, following partial surgical removal, recurred as a muco-epidermoid carcinoma.

Malignant tumors of vascular and neurogenic origin do occur in the mandible but are exceedingly uncommon. Tumors of the marrow or hematopoietic system are more

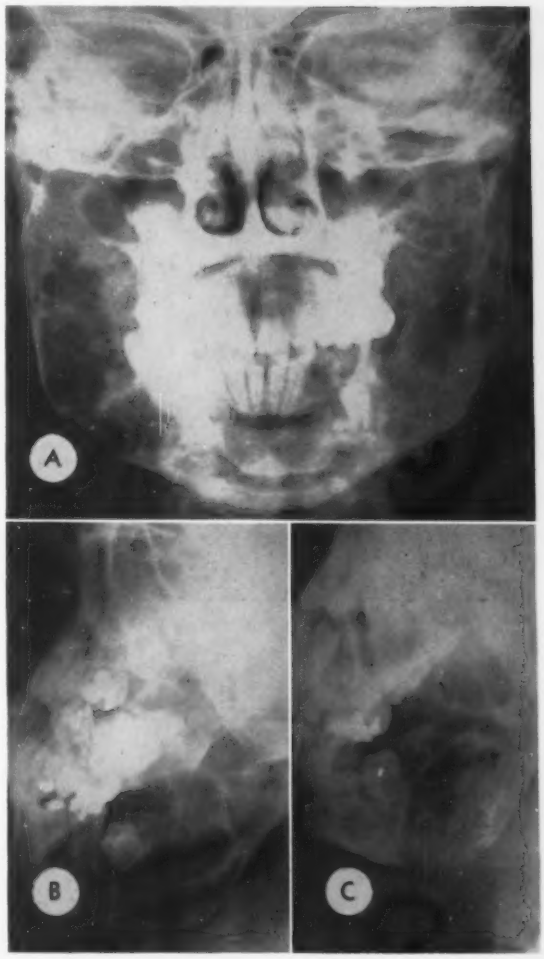

Fig. 16. (*A*, *B* and *C*) A ten year old boy who was never noticed to have mandibular swelling until the local physician called it to the attention of the parents. The patient was asymptomatic and all siblings were normal. The pathologic diagnosis was fibrous dysplasia (cherubism). Note that the involvement is bilateral.

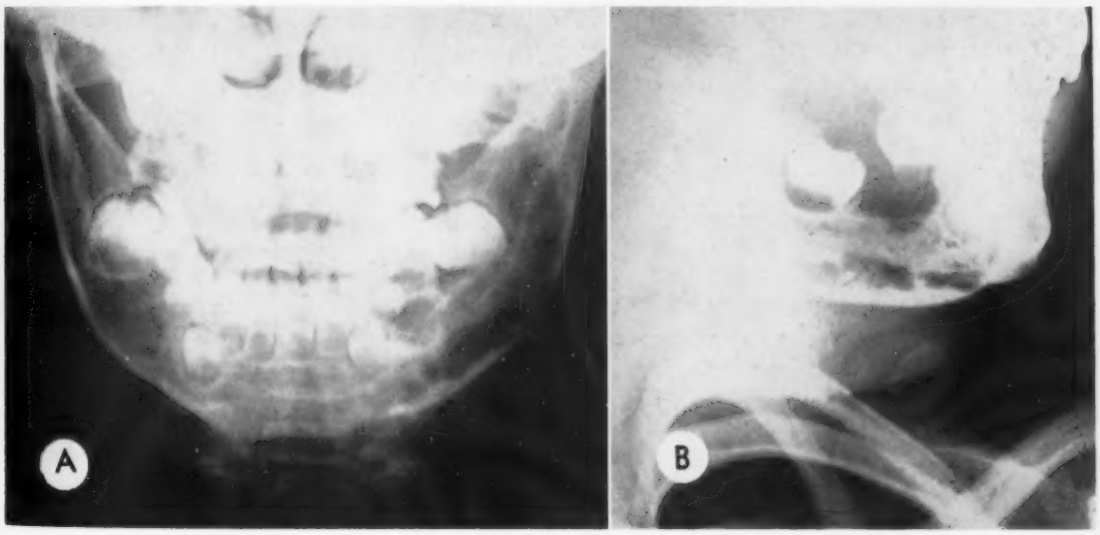

Fig. 17. (*A* and *B*) A three and one-half year old girl was healthy until one year of age when she developed a lump behind the left ear, with an associated yellowish dermatitis of the scalp and diabetes insipidus. Biopsy of the osteolytic lesions of the skull showed Hand-Schül'er-Christian disease.

FIG. 18. A sixteen month old girl had swelling of the left jaw for three weeks which continued to increase in size. The child cried when the area was touched. Biopsy of the mandible showed eosinophilic granuloma. A later biopsy of the skin of the abdomen showed changes compatible with Letterer-Siwe disease.

FIG. 19. A thirty-six year old woman who had pain and aching in the right temporal area for five months at the time this roentgenogram was made. The cyst was curetted and the pathologic diagnosis of the tissue was muco-epidermoid cyst. Five years later the cyst recurred and a biopsy specimen at that time showed muco-epidermoid carcinoma.

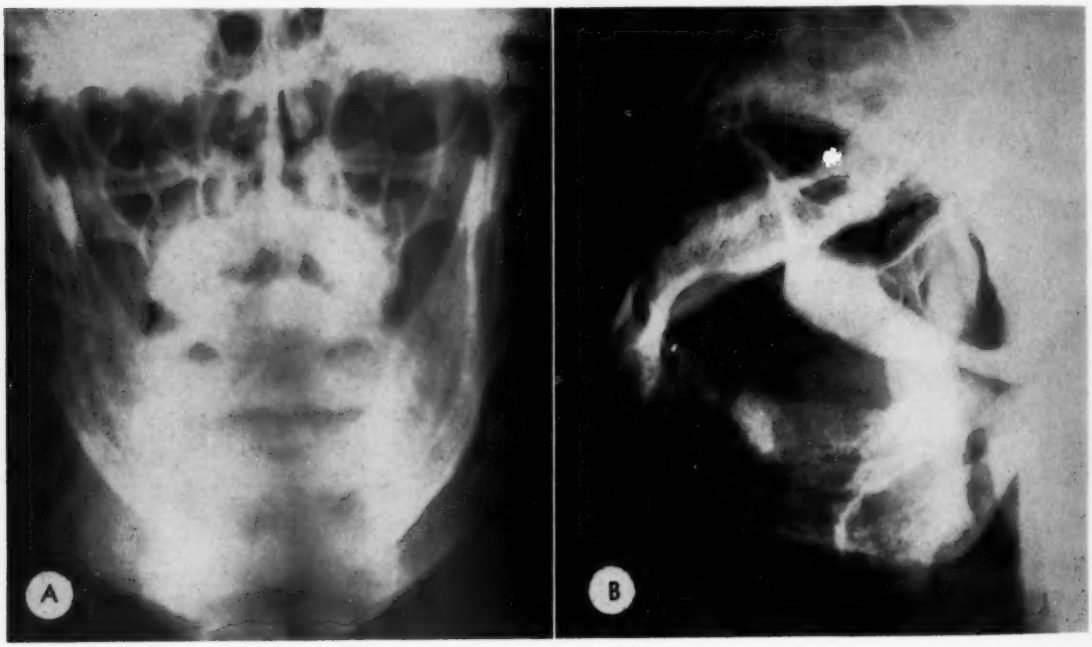

FIG. 20. (*A* and *B*) A forty year old man with pain in the right mandible for one year. Previous treatment consisted of dental extractions and penicillin. The pathologic diagnosis was fibrosarcoma. Note the soft tissue swelling, the fine perpendicular periosteal spicules, and the osteolytic involvement of the body of the mandible.

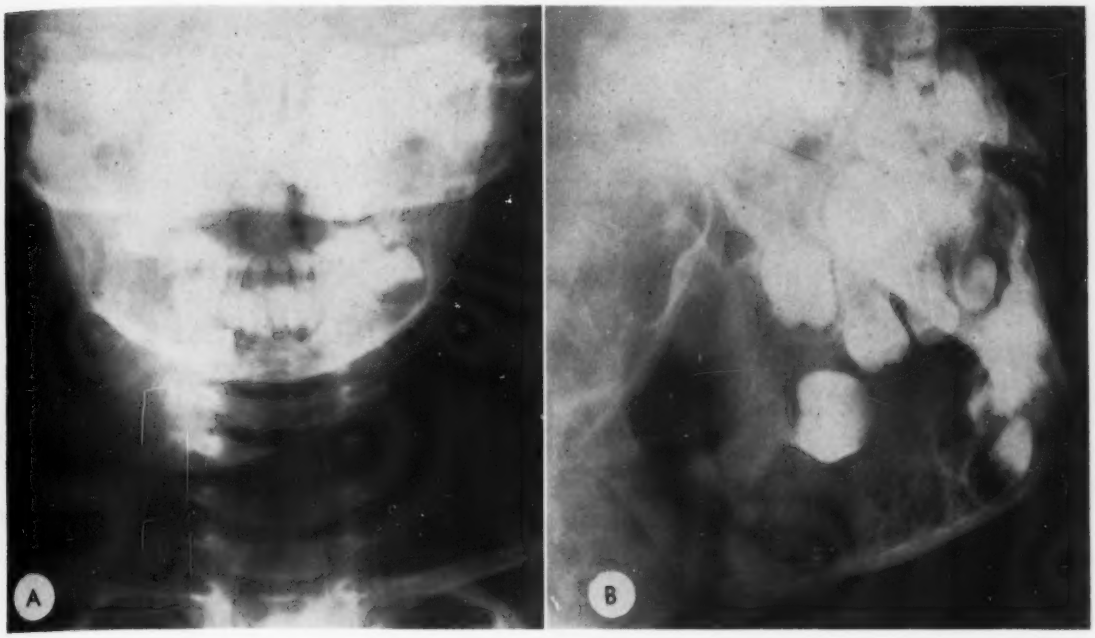

Fig. 21. (*A* and *B*) A three and one-half year old girl had slight swelling over the right lower gum for four weeks which gradually increased in size. Biopsy showed Ewing's sarcoma.

frequent. Because of the embryonic origin of the mandible, sarcomas comprise a large part of the rare primary malignant tumors.

1. *Primary Malignant Tumors.* Primary malignant tumors of the mandible are rare but do occur. Osteogenic sarcoma, Ewing's sarcoma, chondrosarcoma, reticulum cell sarcoma, lymphosarcoma, and fibrosarcoma (Fig. 20, *A* and *B*) are the principal types. As in other bones these tumors produce irregular ill-defined, areas of bone destruction. The margins are indistinct and fade into the surrounding bone (Fig. 21, *A* and *B*). Osteogenic sarcomas[10] and reticulum cell sarcoma may have mixed osteolytic and osteoblastic components.

Osteogenic sarcomas may cause expansion of the cortex with periosteal new bone

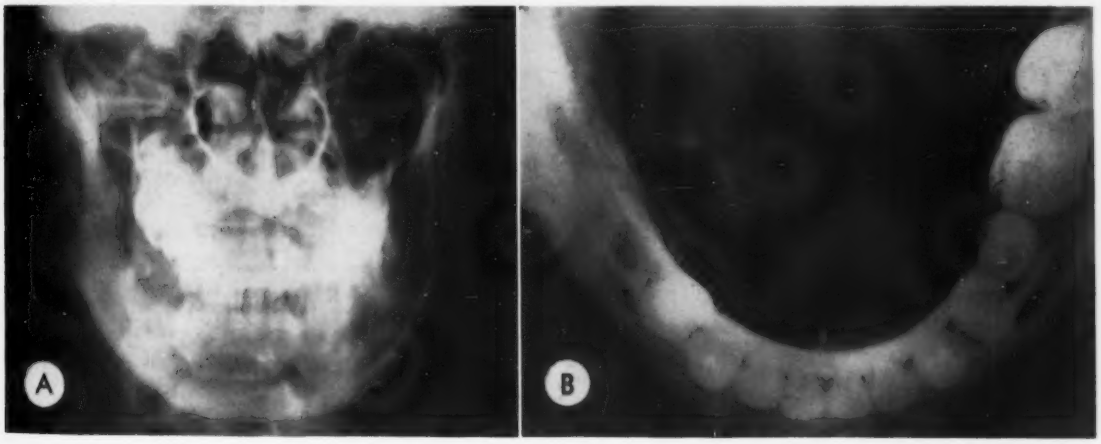

Fig. 22. (*A* and *B*) An eighteen year old woman with a history of a rapidly growing lump on the right lower gum. Just prior to admission she noticed some anesthesia of the lip and teeth. The diagnosis based on biopsy at resection of the mandible was osteogenic sarcoma. Note the soft tissue mass and perpendicular spicules.

Fig. 23. A fifty-four year old man with a firm fixed nodule anterior to the left ear for ten years. Treatment consisted of rubbing with a bland salve and, during the past year, 20 deep radiation treatments were given after a presumptive diagnosis of parotid tumor. Surgical pathologic diagnosis was osteochondrosarcoma.

formation which also occurs in Ewing's sarcoma; however, perpendicular spicules or striations are more common (Fig. 22, *A* and *B*).

Gerry and Williams[4] reviewed the literature in 1955 and found only 6 cases besides the 2 of their own in which the reticulum cell sarcoma was primary in the mandible. In these cases, it was encountered more often in men than in women, and occurred up to the age of forty. All 8 patients had local pain but also had an unusual over-all general feeling of well-being.

Chondrosarcomas are thought to arise from cartilaginous arrests trapped in the mandible[5] or from osteochondromas (Fig. 23). Pain is common and distant metastases occur late.

Multiple myeloma produces a roentgenographic picture of diffuse irregular areas of destruction of bone with erosion of the tooth roots when they are in close proximity. At times, these tumors resemble the more typical "punched out" lesions that occur in involvement of other bones (Fig. 24, *A* and *B*). They are most frequently found in the middle-age group with more men than women affected.[12] Areas of bone destruction without cortication and away from the apex of a tooth should arouse suspicion of this diagnosis. These tumors destroy and perforate the cortex and several small lesions may coalesce to form large lesions. Stafne[15] suggests that when myeloma involves the mandible the cortex will not be expanded.

2. *Metastases.* Metastases to the mandible occur late in malignant diseases, usually after involvement of other bones. The tumors that frequently metastasize to the mandible are those of breast (Fig. 25, *A* and *B*), thyroid, lung, kidney and prostate. Occasionally, head and neck tumors, carcinoma of the gastrointestinal tract, carcinoma of the cervix and vagina, lymphosarcomas and melanomas may metastasize to the mandible.[17]

Roentgenographically, metastases appear as solitary or multiple irregular radiolucent defects. In some tumors, such as those of the prostate or lymphosarcoma, the metastatic area will have a sclerotic component. In any irregular defect with a poorly defined margin, metastasis should be a cardinal differential diagnostic consideration.

3. *Extension from Surrounding Tissues.* Any tumor of the face or oral cavity may extend to involve the mandible. The degree of involvement may vary from a pressure defect in the cortex to actual perforation of the cortex and medullary invasion.

Differential Diagnosis

The final diagnosis of a mandibular lesion should be based upon pathologic examination. Some lesions can be identified by clinical and roentgenographic methods. The rare malignant tumors present such varied features that a pathologic specimen is essential for positive identification.

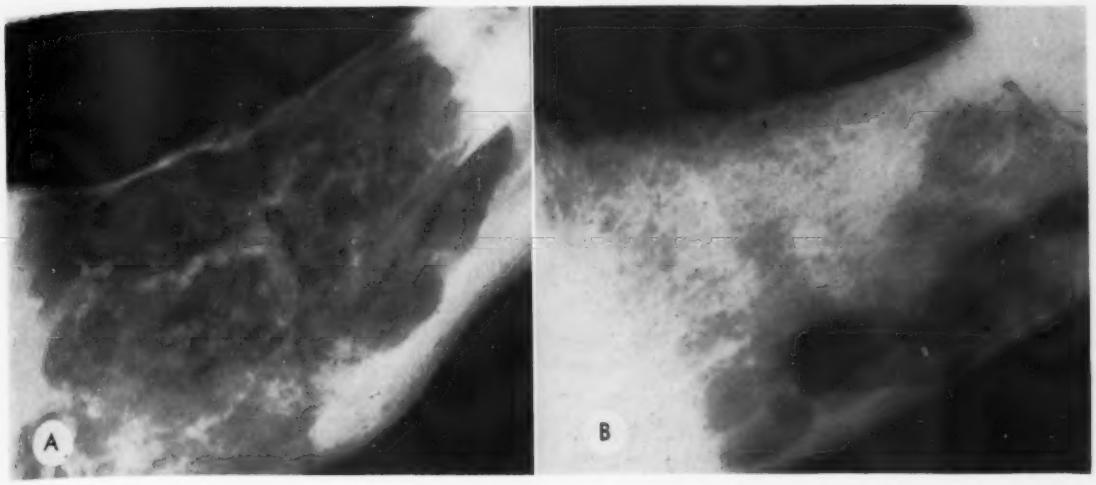

Fig. 24. (*A* and *B*) A sixty-six year old man had rib pain for three months. There were widespread radiolucent defects of bone. Sternal marrow aspirate was diagnostic of multiple myeloma.

To differentiate lesions of the mandible roentgenographically and to classify them tentatively, these questions must be answered and the findings must be correlated:

(1) Is the lesion unilocular, multilocular, or multicystic?

(2) Are the borders of the lesion well defined or diffuse and hazy?

(3) What is the location and extent of involvement?

(4) Is the cortex expanded or destroyed?

(5) Does the involved portion contain calcific or ossifying areas?

(6) Is there periosteal reaction or a soft tissue mass?

(7) Are other bones involved?

A single cystic lesion with a well-defined margin that has no relationship to a tooth is most frequently a squamous cyst. If it is oriented to the root of a tooth, then the

Fig. 25. (*A* and *B*) A thirty-three year old woman who had a right radical mastectomy for scirrhous carcinoma of the breast. Note the complete destruction of the cortex by an osteolytic process.

possibility of a radicular cyst should be considered. When a cystic lesion is oriented to the crown or contains a tooth, it is probably a dentigerous cyst. Fibromas and myxomas are tumors that may simulate dentigerous or radicular cysts. Infrequently, fibrous dysplasia may be seen roentgenographically as a single monolocular lesion. Neurofibromas are observed as an oval radiolucency aligned to the central mandibular canal or in the subperiosteal area.

Squamous cysts may have a multilocular well-defined appearance on initial roentgenographic study, but additional roentgenography will often reveal a single cyst with lobulated margins.

Fibrous dysplasia commonly has a multilocular appearance with gross expansion of the cortex; the "smudged" appearance due to osseous metaplasia is characteristic if present. Fibrous dysplasia is frequently unilateral while cherubism is bilateral and occurs in a younger age group.

A difficult differential diagnostic problem arises when there are multiple well-defined cystic areas. Ameloblastomas have varied appearances but the typical one is of multiple small radiolucencies arranged in discrete clusters, like grapes. The compartments are usually rounder than those of the giant cell tumors, and the trabeculae of the giant cell reparative granulomas or of the tumors of hyperparathyroidism are more numerous and not as distinct as those of ameloblastomas. To distinguish between giant cell granulomas and hyperparathyroidism, it is necessary to review the clinical and laboratory data. Defects of reticuloendotheliosis do not occur in clusters and rarely affect *only* the mandible. Their most characteristic location is in the alveolar process with destruction and the appearance of "floating teeth."

Single or multiple radiolucent defects with poorly defined margins should always suggest metastases (Fig. 23). It is here that care should be taken to differentiate between metastases, myeloma, and primary malignant tumors. Biopsy is indicated if the diagnosis is not conclusive from the clinical and laboratory data. Primary malignant tumors may mimic any lesion but the roentgenographic characteristics of malignant tumors of the mandible are the same as of other bones. Irregular osteolytic or osteosclerotic areas with periosteal reaction are typical.

The odontogenic tumors show areas of calcification surrounded by a radiolucent area of varying thickness which indicates encapsulation. If the calcifications are grossly irregular or toothlike, the tumor is an odontoma. Cementomas are attached to a tooth root while enamelomas are more radiopaque and appear at the bifurcation of the tooth roots.

Osteomas of the mandible are rare; they usually occur at the angle of the mandible and roentgenographically appear as a dense tumor on a pedicle. Osteoid osteomas have the typical appearance of a sclerotic area with a central nidus. Infantile cortical hyperostosis is diagnosed on the basis of the age of the patient, his clinical history, and the roentgenographic finding of thickened lamellated subperiosteal bone. Infrequently, fibrous dysplasia may appear roentgenographically as a dense irregular sclerosis with indistinct margins.

Tumors that involve the mandible by direct extension from the surrounding tissues are usually recognized clinically and the mandible is examined roentgenographically to confirm bone involvement. In some rare borderline cases, however, a biopsy must be performed to distinguish between a mandibular tumor with soft tissue extension and a primary soft tissue tumor with mandibular involvement.

While not included in this discussion, osteomyelitis and radiation necrosis should be considered in the differential diagnosis, although the clinical history will usually eliminate these entities.

CONCLUSION

1. Some cysts and tumors may be diagnosed from the roentgenograms with reasonable accuracy.

2. In most cases, however, a biopsy is almost mandatory because of the extremely variable roentgenographic features of mandibular cysts and tumors.

3. Roentgenographic findings of mandibular cysts and tumors are presented to aid in the differential diagnosis.

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THE RELATIONSHIP OF FASTING BLOOD GLUCOSE TO ORAL ROENTGENOGRAPHIC FINDINGS*

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A SURVEY of the literature shows a tendency of many investigators to associate the roentgenographic findings of alveolar bone loss and periodontal widening with the diabetic patient. There is implication of a linear relationship between these two findings and the diabetic state. In other words, the more pronounced the diabetic state (the higher the blood glucose) the more severe the alveolar bone loss and/or periodontal widening.

One report[1] has already analyzed the relation of oral roentgenographic findings with the glucose tolerance pattern. The evidence from that study indicates that there is a relationship between the glucose tolerance pattern and the presence of alveolar bone loss and periodontal widening.

This report will analyze the relationship of the two roentgenographic signs (*alveolar bone loss* and *periodontal widening*) to the fasting blood true glucose in a group of 100 routine dental patients.

REVIEW OF THE LITERATURE

Published material is available regarding the relationship between diabetes mellitus and alveolar bone loss and periodontal widening.

Alveolar Bone Loss. Many investigators have reported this finding as being associated with the diabetic condition.[4,5,8,10–12,18] One investigator[14] observed bone loss in 1 out of every 2 of a group of diabetic children. However, two teams[6,13] claim no alveolar bone loss. Sheridan *et al.*[15] observed normal alveolar bone in 24 per cent of patients with normal glucose tolerance patterns. They further observed that patients with increased or decreased glucose tolerance showed a greater incidence of alveolar bone loss and that most of the diabetic patients showed considerable bone loss in contrast to other groups of patients having more physiologic glucose tolerance patterns.

Periodontal Widening. Rutledge[14] observed this finding in 14 of 20 juvenile diabetics. The observations of Rudy and Cohen[13] on this finding are in agreement with those of Rutledge. However, Ziskin *et al.*[19] could not demonstrate periodontal widening in their series of cases. Sheridan *et al.*[15] reported that in patients with decreased glucose tolerance patterns, *i.e.*, persons who would commonly be called diabetic, there was a 62 per cent incidence of periodontal widening. These investigators also observed that the occurrence of this finding decreased with an increase in sugar tolerance.

METHOD OF INVESTIGATION

One hundred dental patients were studied in the Section on Oral Medicine at the University of Alabama School of Dentistry. As far as possible, the subjects were selected at random. Table 1 shows the age distribution of the group. It can be observed that the pattern approaches a typical unimodal curve. Included in this study were 22 males and 78 females.

The roentgenograms of each patient were studied for possible alveolar bone loss and periodontal widening. Alveolar bone loss was scored on a seven point scoring system where zero was the score given for a normal septal crest; a score of one was assigned for minimal alveolar bone loss; two for minimal to moderate alveolar bone loss; three for one-half the alveolar support missing; four for moderate to severe alveolar bone loss; five for no alveolar support;

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and six for edentulous cases. Periodontal widening was scored on a four point scoring system where zero was the score given for no marginal widening; a score of one was given for minimal marginal widening; two for severe marginal widening; and three for edentulous cases.

A true glucose tolerance test was performed on each patient according to the methods of Somogyi[16,17] and Nelson.[9] No preparatory diet was recommended except for complete fasting for twelve hours prior to the laboratory examination. Complete details regarding the method of investigation are available in the publication by Sheridan *et al.*[15] For this study, only the information derived from the fasting blood glucose measurements was utilized.

RESULTS

The fasting blood glucose determination will be considered in the light of: (A) single roentgenographic signs, and (B) a combination of the two signs.

A. SINGLE SIGNS

An analysis of the data will be offered for: alveolar bone loss, and periodontal widening.

Alveolar Bone Loss. This roentgenographic sign will be discussed in terms of: (1) mean scores, (2) coefficients of variation, and (3) statistical significance. The scores of edentulous cases were included in this analysis since this condition would be an end result of alveolar bone loss. Although loss of teeth can be due to other causes, it is felt that the exclusion of these cases would be too discriminatory against those cases where the loss of teeth was due to alveolar bone loss. The available data do not permit the selection of those cases where loss of teeth was accompanied by severe alveolar bone loss.

TABLE I

AGE DISTRIBUTION

Age Group	Total No. of Patients
0–9	1
10–19	9
20–29	19
30–39	27
40–49	19
50–59	17
60–69	8
Total	100

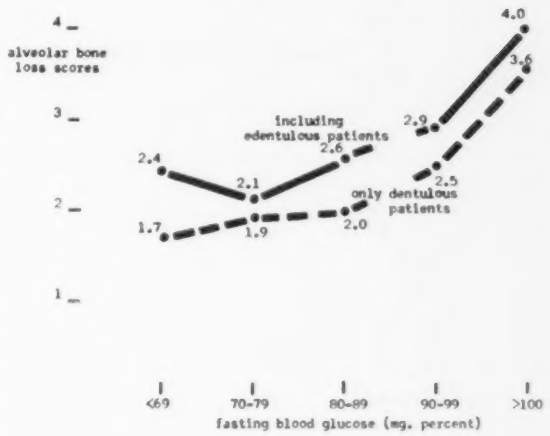

FIG. 1. Relationship of fasting blood glucose to alveolar bone loss.

1. Mean Scores. An examination of Figure 1 shows that alveolar bone loss was least in those patients with a fasting blood glucose of 70–79 mg. per cent. There was an increase in the scores calculated from patients having a fasting blood glucose of <69, 80–89, and 90–99 mg. per cent. There was a marked increase in alveolar bone loss in those patients with a fasting blood glucose of >100 mg. per cent. Figure 1 also shows a comparison with the mean scores obtained from dentulous cases alone. Basically, the curves follow the same pattern except in the <69 mg. per cent group. When the edentulous cases are excluded from this group, the mean score for alveolar bone loss is slightly less than the 70–79 mg. per cent group.

2. Coefficients of Variation. An examination of Table II reveals that basically the coefficients of variation decrease with an increase in the blood glucose percentages. The conclusion to be drawn is that the individual subjects seem to cluster about

TABLE II

RELATIONSHIP OF FASTING BLOOD GLUCOSE TO ALVEOLAR BONE LOSS

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Mean Alveolar Bone Loss Score	2.4	2.1	2.6	2.9	4.0
Standard Deviation	2.0	1.5	1.9	1.8	1.1
Coefficient of Variation	83	71	73	62	28
Mean Difference	0.3	0.5	0.3	1.1	
Standard Error of Difference	0.5	0.4	0.7	0.8	
Observed *t* Value	0.60	1.25	0.43	1.38	
Degrees of Freedom	50	71	43	15	
P	.5	>.1	.5	>.1	

the mean more in the upper blood glucose groups than in the lower ones.

3. Statistical Significance. From Table II it can be seen that there is no statistically significant difference between any of the blood glucose groups when compared to similar groups that are greater or less by 10 mg. per cent. This is evidenced by the probability factor of .5 and >.1. The P value was also checked for all the groups. There were no statistically significant differences shown except those between the >100 mg. per cent group and the <69, 70–79, and 80–89 mg. per cent groups. There the P values were <.05, <.005, and <.025, respectively. It is evident that the greatest significant difference occurs between the >100 mg. per cent group and the 70–79 mg. per cent group.

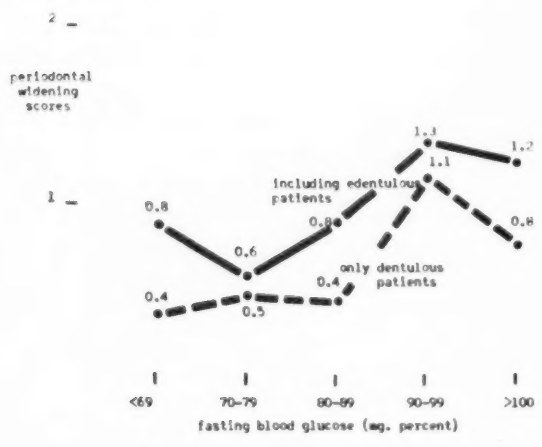

FIG. 2. Relationship of fasting blood glucose to periodontal widening.

Periodontal Widening. This roentgenographic sign will be discussed in terms of (1) mean scores, (2) coefficients of variation, and (3) statistical significance.

1. Mean Scores. Figure 2 shows the mean scores of all patients (dentulous and edentulous) and the mean scores when edentulous patients were excluded. An examination of these scores shows the effect of excluding the edentulous cases. Patients having severe periodontal widening would be scored but once, having lost their teeth, would not be counted and thus would lower the mean score of the blood glucose group they belong to. This effect is greatest in the <69 and >100 mg. per cent blood glucose groups. The rest of this analysis will pertain to the scores obtained from the study of all the patients. Figure 2 shows that periodontal widening is least in the 70–79 mg. per cent blood glucose group and, basically, this condition increases as the fasting blood glucose rises above or falls below that of this group.

2. Coefficients of Variation. An examination of Table III shows that the smallest coefficient of variation occurs at the 90–99 mg. per cent group and is greatest at the <69 group. In other words, the individual scores more closely cluster about the mean in the 90–99 mg. per cent group.

3. Statistical Significance. From Table

TABLE III

RELATIONSHIP OF FASTING BLOOD GLUCOSE TO PERIODONTAL WIDENING

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Mean Periodontal Widening Score	0.8	0.6	0.8	1.3	1.2
Standard Deviation	1.1	0.8	1.0	0.7	1.2
Coefficient of Variation	138	133	125	54	100
Mean Difference	0.2	0.2	0.5	0.1	
Standard Error of Difference	0.32	0.22	0.28	0.54	
Observed *t* Value	0.63	0.91	1.79	0.19	
Degrees of Freedom	48	69	41	13	
P	.5	>.2	>.05	.5	

III it can be seen that there is no statistically significant difference between any of the blood glucose groups when compared to similar groups that are greater or lesser by 10 mg. per cent of blood glucose. This is evidenced by probability values that are greater than .05. The P value that most closely approaches significance is shown between the 80–89 and 90–99 mg. per cent groups. The P value was also checked for all the groups. There were no statistical differences shown except when the 70–79 and 90–99 groups were compared. A P value of <.005 was obtained when the scores of these two groups were analyzed.

B. COMBINATION OF THE TWO ROENTGENOGRAPHIC SIGNS

An analysis of the data will be offered for alveolar bone loss and periodontal widening. The combination of these two signs will be discussed in terms of (1) mean scores, (2) coefficients of variation, and (3) statistical significance. There exists a difference in the scoring systems of these two signs, alveolar bone loss being measured on a six point scale and periodontal widening on a three point scale. In order to equate these scores, the periodontal scores were doubled so that a value could be placed on the combination of these two roentgenographic signs.

1. Mean Scores. It appears from Figure 3 that the relationship between fasting blood glucose and the presence of these two signs is parabolic. In other words, alveolar bone loss and periodontal widening are least with fasting blood levels in the 70–79 mg. per cent range. As the fasting blood glucose increases or decreases, the combination of alveolar bone loss and periodontal widening increases.

2. Coefficients of Variation. Table IV shows that the coefficients of variation more or less decrease with an increase in the blood glucose groups. The conclusion to be drawn is that the individual findings more

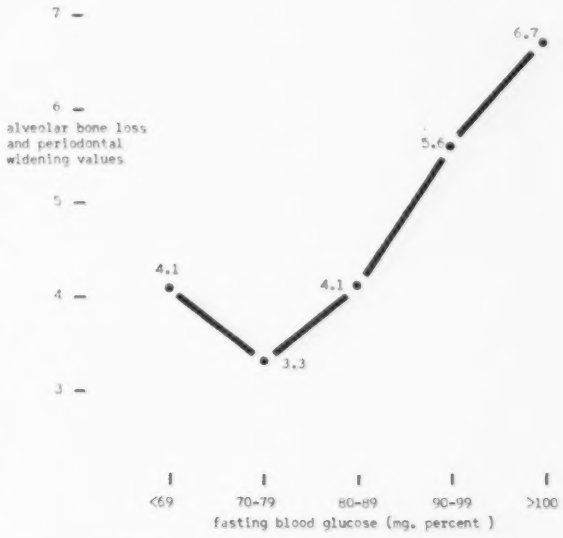

FIG. 3. Relationship of fasting blood glucose to alveolar bone loss and periodontal widening (100 routine dental patients).

TABLE IV

RELATIONSHIP OF FASTING BLOOD GLUCOSE TO ALVEOLAR BONE LOSS AND PERIODONTAL WIDENING

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Mean Alveolar Bone Loss and Periodontal Widening Scores	4.1	3.3	4.1	5.6	6.7
Standard Deviation	4.2	2.9	3.8	3.0	2.7
Coefficient of Variation	102	88	93	54	40
Mean Difference	0.8	0.8	1.5	1.1	
Standard Error of Difference	1.30	0.86	1.22	1.49	
Observed *t* Value	0.615	0.930	1.229	0.738	
Degrees of Freedom	48	69	41	13	
P	.5	>.2	>.2	>.4	

closely cluster about the mean in those cases with hyperglycemia.

3. Statistical Significance. Table IV shows no statistically significant difference between any of the fasting blood glucose groups and their adjacent group or groups. A comparison of each group with all others showed no statistically significant differences except between the 70–79 mg. per cent group, and the 90–99 and >100 mg. per cent groups. The P values here were <.05 and <.01, respectively.

DISCUSSION

The data will be discussed in the following fashion: (1) mean scores, (2) coefficients of variation, and (3) significant probability values. Because of the close correlation between age versus glucose levels and roentgenographic signs versus age, the age factor will be analyzed in terms of blood glucose and roentgenographic signs.

1. *Mean Scores.* An analysis of the mean score values for the two roentgenographic signs, considered independently and in combination, points up the fact that the patterns are parabolic. Moreover, the parabolic picture is more clearcut when the two signs are combined. These data would tend to indicate that there is a range of blood glucose normality for a particular roentgenographic sign or group of signs (Table V) in the general range of 70–79 mg. per cent. This information also suggests that fasting blood glucose levels above and below these narrow ranges are apt to be associated with roentgenographic findings.

2. *Coefficients of Variation.* Generally speaking, the lower the coefficient of varia-

TABLE V

COMPARISON OF MEAN SCORES

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Alveolar Bone Loss	2.4	2.1	2.6	2.9	4.0
Periodontal Widening	0.8	0.6	0.8	1.3	1.2
Alveolar Bone Loss and Periodontal Widening	4.1	3.3	4.1	5.6	6.7

TABLE VI

COMPARISON OF COEFFICIENTS OF VARIATION

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Alveolar Bone Loss	83	71	73	62	28
Periodontal Widening	138	138	125	54	100
Alveolar Bone Loss and Periodontal Widening	102	88	93	54	40

tion, the more the individual scores cluster about the mean. Thus, a coefficient of variation of zero is the ultimate in that it signifies that all subjects provide scores exactly like the mean.

Table VI summarizes the coefficients of variation with respect to the fasting blood glucose groups and the single and combined roentgenographic signs. It can be seen that basically the individual scores cluster more closely in the higher blood glucose groups.

3. *Significant Probability Factors.* Table VII gives a tabulation of the significant probability findings for the two single and combined roentgenographic signs. It is noteworthy that patients having fasting blood glucose levels of >100 mg. per cent show significant differences in the amount of alveolar bone loss when compared to those with levels below 90 mg. per cent. For periodontal widening, the only significant differences occurred when the 70–79 and 90–99 mg. per cent groups were compared. However, significant differences appeared between the 70–79 and 90–99 mg. per cent groups, and the 70–79 and >100 mg. per cent groups, when the two roentgenographic signs were combined. It is clear that the greater the hyperglycemia, the more significant is the difference in these combined roentgenographic signs, when these patients are compared with those having blood glucose levels of 70–79 mg. per cent.

AGE FACTOR

There are some investigators who contend that with increasing age the normal blood glucose values increase. Certainly, the data from which this study is derived indicate that roentgenographic signs, in the main, occur predominantly in the older age groups (Table VIII). Hence, it was thought necessary to analyze the relationship between roentgenographic signs and blood glucose levels in a single age group.

Figure 4 graphically demonstrates the relationship of fasting blood glucose to alveolar bone loss and periodontal widening in the 19 patients between the ages of forty

TABLE VII

COMPARISON OF SIGNIFICANT PROBABILITY VALUES

	Relationship of the Fasting Blood Glucose Groups (mg. per cent)	P Value
Alveolar Bone Loss	<69 with >100	<.050
	70–79 with >100	<.005
	80–89 with >100	<.025
Periodontal Widening	70–79 with 90–99	<.005
Combination of Alveolar	70–79 with 90–99	<.050
Bone Loss and Periodontal Widening	70–79 with >100	<.010

TABLE VIII

PERCENTAGE FREQUENCY OF PATIENTS WITH ROENTGENOGRAPHIC SIGNS BY AGE GROUPS

Age Group	Alveolar Bone Loss	Periodontal Widening	Alveolar Bone Loss and Periodontal Widening
0–9	0	0	0
10–19	44	22	11
20–29	78	39	39
30–39	96	52	52
40–49	100	74	74
50–59	100	50	50
60–69	100	86	86

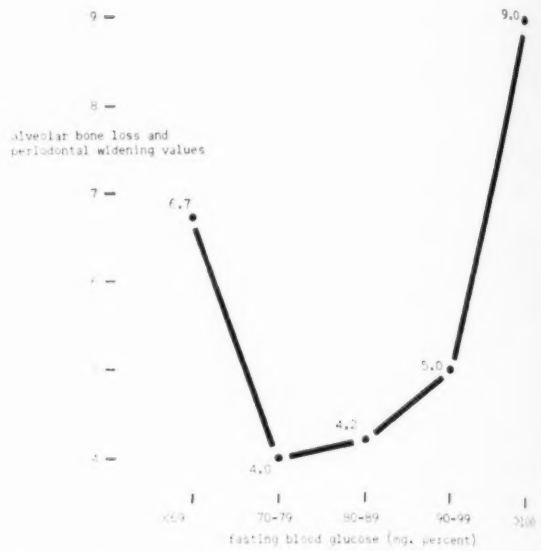

FIG. 4. Relationship of fasting blood glucose to alveolar bone loss and periodontal widening in patients forty to forty-nine years of age.

and forty-nine. An examination of Figure 4 and a comparison between these data and the findings in Figure 3 indicates that the parabola is more exaggerated when the age factor is eliminated. There were no significant differences observed when each group was compared to any other group (Table IX). However, a comparison of Tables IV and IX shows that the individual scores for the one age group cluster more about the mean than was observed for the entire group as judged by the coefficients of variation. Thus it appears that age, or some factor operating with age, may be involved in the relationship of the two roentgenographic signs and glucose metabolism.

SUMMARY

1. This report analyzes the relationship of two roentgenographic signs (*alveolar bone loss* and *periodontal widening*) to the fasting blood true glucose in a group of 100 routine dental patients.

2. An examination of the available data points out the need for evaluating edentulous patients as to the cause of loss of their teeth.

3. An analysis of the mean score values

TABLE IX

RELATIONSHIP OF FASTING BLOOD GLUCOSE TO ALVEOLAR BONE LOSS AND PERIODONTAL WIDENING (40–49 YEAR AGE GROUP)

	Blood Glucose Groups (mg. per cent)				
	<69	70–79	80–89	90–99	>100
Mean Alveolar Bone Loss and Periodontal Widening Values	6.7	4.0	4.2	5.0	9.0
Standard Deviation	4.8	1.4	2.0	0.0	4.2
Coefficient of Variation	72	35	48	0	47
Mean Difference	2.7	0.2	0.8	4.0	
Standard Error of Difference	2.87	1.06	0.91	3.00	
Observed *t* Value	0.94	0.19	0.88	1.33	
Degrees of Freedom	8	10	5	2	
P	>.4	.5	>.4	>.2	

for the two signs, considered independently and when combined, points up the fact that the patterns are generally parabolic. Moreover, the parabolic pattern is more clearcut when the signs are combined than when each sign is analyzed alone.

4. A study of the coefficients of variation shows that, in general, the individual scores cluster more closely about the mean in the higher blood glucose groups.

5. An analysis of the significant probability values shows that the 70–79 mg. per cent fasting blood glucose group has P values of $<.05$ when compared to one of the higher groups for the roentgenographic signs, singly and in combination.

6. The findings in this report suggest that the two roentgenographic signs (alveolar bone loss and periodontal widening) are associated with both hyper- and hypoglycemia.

7. The evidence derived in this report suggests that the age factor, or some other factor operating with age, is associated with hyper- and hypoglycemia in the relationship of these two roentgenographic signs and glucose metabolism.

8. These data suggest that the range of fasting blood glucose normality may be more narrow than is usually accepted.

9. Earlier reports[2,3] have shown that the two-hour blood glucose is a better indicator of glucose metabolism than fasting blood glucose with regard to oral symptoms. A report to follow[7] will analyze the relationship of the two-hour blood glucose to these roentgenographic signs.

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BILATERAL BENIGN BONE CYST OF THE OS CALCIS*

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THE os calcis can be the site of any type of osseous or other connective tissue pathology. As far as can be determined from the literature, the os calcis is neither exempt from nor more prone to any particular lesion. Among lesions described involving the os calcis, benign bone cysts have been sporadically reported.[1–8] In most of the cases mentioned, the cyst was discovered as an incidental finding when the extremity was roentgenographed following trauma. The cysts occurred in the anterior inferior portion of the bone. This is the classic site given in all reports. We would like to describe a case of a benign bone cyst which was found in an unusual location.

REPORT OF A CASE

The patient was a fifty-one year old white female with a congenital kyphoscoliosis who had sustained an injury to her right foot and ankle in a fall. Immediately after the fall there were pain and swelling of the heel and ankle. Weight could not be borne on the affected extremity. There was no history of trauma or disability of the lower extremity previous to the injury. There was no known background of a metabolic or endocrine disturbance. A roentgenogram of the right foot and ankle after admission to the University Hospital revealed a comminuted fracture of the os calcis (Fig. 1). The fracture had occurred at the posterior superior aspect of the bone. The fracture fragments appeared to have composed the posterior superior wall of a cystic lesion.

Because of the unknown nature of the cystic lesion and because of the displaced bone fragments and partial Achilles tendon avulsion, surgical exploration was indicated. At surgery, five days after the trauma, a large unicameral cyst was found. This was roughly ovoid in form with a small, globular outpocketing anteromedially. The cyst contained small blood clots and a moderate amount of serosanguinous fluid. A very thin lining membrane was disclosed through curettage. The specimen submitted to the Pathology Laboratory consisted of fragments of the fractured os calcis with the contents of the curetted cystic area. The bone specimen showed a fairly acellular layer of fibrous tissue lining some parts of the surface of the bony fragments. This fibrous tissue in places revealed evidence of hemorrhage. The curetted specimen consisted of dense fibrous tissue and clot. The pathologic diagnosis was "fragments of bone cyst."

The laboratory tests included a complete blood count, urinalysis and sedimentation rate, serum calcium, phosphorus, phosphatase (acid and alkaline), total proteins, A-G ratio and creatinine, urinary creatinine, calcium and phosphorus, and tests for tubular resorption of phosphorus. The findings were all within normal limits.

Figure 2 shows the right ankle after surgical exploration and removal of the comminuted fragments. For purposes of comparison, a roentgenographic study of the left os calcis was done (Fig. 3 and 4). Surprisingly, this also revealed a triangular area of radiolucency at the posterior superior portion of the os calcis. There was an apparent horizontal septal division inferiorly. The lesion was marginated by a line of increased bone density. Bone trabeculation and density were normal throughout the remainder of the os calcis and visualized extremity. We assumed this to represent a benign cyst of the opposite os calcis. A skeletal survey was performed to rule out other cystic lesions. None were found. The patient's postoperative period was uneventful.

DISCUSSION

A major problem confronting the roentgenologist on the demonstration of a bone lesion is to decide whether it is benign or malignant. The expression of this potential

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Fig. 1. Roentgenogram of the right ankle showing comminuted fragments, displaced superiorly, which appear to have composed the posterior superior wall of a cystic lesion.

Fig. 3. Roentgenogram of the left ankle demonstrating a radiolucent lesion with a densely marginated border in the posterior superior portion of the os calcis.

Fig. 4. Roentgenogram of the left os calcis in a sagittal view.

Fig. 2. The right ankle after surgical exploration and removal of the comminuted fragments.

is the same for the os calcis as for other areas of the osseous system. A sharply delineated lesion with a discrete condensed border points to the benign nature of the abnormality. It is possible to have a sharply delineated lesion in a malignant process, as in a myeloma. It is also possible to have a sharply delineated lesion in a benign process such as gout or an eosinophilic granuloma. However, the presence of a densely marginated border almost always indicates a benign entity. When such a lesion which does not demonstrate significant expansion from within is presented, it reinforces the impression of benignity. Laboratory data are important in confirming the roentgenographic impression.

We believe that cysts of the os calcis are probably of greater frequency than the relatively few reports would lead one to suppose. The available literature does not mention the possibility of a contralateral lesion. The fact that the lesion in this case is presumed to be bilateral (the contralateral side was not biopsied) and that it is not in the accepted classic location does not detract from the diagnosis of a benign bone cyst.

Benign bone cysts can be found in any part of the skeletal system derived from endochondral bone. These lesions occur near an epiphyseal line and by juxtaepiphyseal growth migrate toward the diaphysis. The basic formation of the cyst comes about through a limited disorder of an early member of the osteogenic sequence rather than through disturbance of a mature bone element. We used the term "limited" above because, once the cyst is formed, normal bone formation and growth may occur at the site from which the cyst arose. This explains the normal bone intervening between the cyst and the epiphyseal line. The relatively dense surrounding border may represent a proliferative response to the lining membrane; it may result from the pressure of the enclosed fluid or from diffusion of osteogenic inductive elements from the fluid or membrane. If this border were purely a mechanically induced phenomenon caused by diversion of stress lines, one would expect, in an eccentrically placed lesion in a long bone, marked asymmetry of the margin; where the border parallels the main force lines, it should be thicker. In general, the bone density surrounding the cyst bears no relationship to the axial stress configuration, so the border apparently is not simply a mechanically induced phenomenon.

SUMMARY

Bone cysts of the os calcis have been described in the literature. Previous authors have stressed the localization of a benign cyst to the anterior inferior portion of the os calcis. A case has been reported in which the lesion occurred at the posterior superior portion of the os calcis with the roentgenographic demonstration of a similar lesion in the contralateral bone.

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THE ROENTGENOLOGIC FEATURES OF ACROMEGALY

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THE advanced stages of acromegaly show characteristic roentgenologic changes of the bones and joints. Particularly, the skull roentgenograms may prove to be of diagnostic importance. In most cases, there is enlargement and erosion of the sella caused by the increasing pressure of the hyperplastic pituitary gland. The roentgenologic manifestations in other structures occur rather late in the course of the disease; nevertheless, these changes are of great importance for the understanding of the dynamics and pathogenesis of this disease.

PATHOGENESIS

It is well established that the disease is due to an increase in function and size of a portion of the anterior lobe of the pituitary gland.[12] Pathologically, the lesion is most commonly an eosinophilic adenoma but, occasionally, mere hyperplasia of the eosinophilic cells without actual adenoma formation or even eosinophilic adenocarcinoma or chromophobe adenoma may give rise to this clinical syndrome. The expansile growth of the pituitary neoplasm causes the familiar erosion of the osseous wall of the sella and the characteristic bulging of the diaphragm. The absence of roentgenologic changes of the sella, however, does not allow one to exclude the diagnosis of acromegaly, since the pituitary gland may show marked increased hormonal activity without significant enlargement and hyperplasia. The most obvious manifestations of the disease are caused by the liberation of excessive amounts of growth hormone. The mere pressure of the tumor, however, may result in a disturbance of the other pituitary functions of the uninvolved portion of the gland or may cause symptoms by pressure on adjacent nerves.[4]

CLINICAL AND ROENTGEN FEATURES

The excessive amount of growth hormone or somatropin has a direct effect on the osseous, cartilaginous and connective tissues.[9,10,11,21] In childhood, the overproduction of growth hormones causes so-called pituitary giantism. If the onset of the disease is during early adolescence, giantism with acromegalic features is the usual form of presentation. If, however, the onset of the disease is in adulthood, one observes the typical acromegalic form.[1,2,3]

The accentuation of normal appositional growth of bone is the most significant roentgenologic feature of this disease. Marked spur formation and relatively early appearance of degenerative osteoarthritic changes are noted in the majority of patients.[7,8] This appositional bone growth is most characteristically demonstrated in areas of normal bone prominence such as tuberosities and sites of muscular or ligamentous attachment.[8] Periosteal new bone formation, particularly along the anterior margins of the vertebral bodies, is one other classic manifestation of this type of process.[6,10,21] The typical facial appearance of the patient is caused by the prominence of the zygomatic arches and supraorbital ridges, as well as by prognathism. These changes again are due to appositional bone growth as well as overgrowth and elongation of the mandible and lower teeth.

Elongation of the bones by endochondral growth is seen more rarely than appositional growth, mainly because most patients are observed during puberty or later, when most epiphyses have fused. It is stated, however, that even in this age group there may be limited endochondral growth at special sites.[18,24] Weinberg points out that, particularly in the deep layers of the articular cartilage, there may be endo-

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chondral growth resulting in increasing length of the tubular bones, even after what appears to be complete obliteration of the epiphyseal cartilage. Marked cartilage proliferation, however, is seen in the regions of the costochondral junctions, as well as in the joints of the hands and feet.[18,23] The costochondral junction of the ribs shows marked proliferation below both the perichondrium and the periosteum. This results in marked prominence of this region, thereby causing the so-called acromegalic rosary. A marked proliferation of joint cartilage has long been recognized as a classic feature of this disease.[7,9,20,23] The nature of the rapidly formed cartilage, however, appears to be in considerable doubt. Early degenerative changes are found in periarticular areas, and particularly so in the region of the junction of the perichondrium and periosteum.

The collagen tissue also shows a marked response to the excessive amount of growth hormone. Thickening of the skin, increase of the periportal connective tissues, increase of the supporting network of the bronchi, changes in the connective tissues supporting the renal parenchyma, and even interstitial tissue hypertrophy of the pancreas have been reported.[8,14,16] There may be painful swelling of the finger joints and generalized enlargement of the soft tissues of the fingers, as well as actual thickening of the synovia as seen on microscopic sections.

Generalized and localized osteoporosis is a well-known feature of this disease.[7,18,19,22] The most typical example of localized resorption of bone is the classic enlargement of the frontal sinuses. This is undoubtedly caused by a dual process, *i.e.*, both bone resorption and apposition, resulting in a large frontal sinus with bulging of the supraorbital ridges. The tubular bones are consistently subjected to both resorption and apposition. Curschmann[7] and Kellgren *et al.*[18] have emphasized the excessive modeling of tubular bones resulting in abnormally thin, spindly phalanges.[7,18]

ROENTGEN FINDINGS

The material analyzed for this paper consists of 31 patients, most of them with long-standing symptoms (mean duration of disease, nine years). The age distribution ranged between twenty-four and seventy-seven years. The roentgen findings are tabulated in Tables I and II.

SKULL

The classic changes of the sella were encountered in 29 of 31 patients (Table I). Only 2 patients showed a sella which was considered normal in size and contour. The most frequently encountered manifestation was generalized enlargement of the sella with rarefaction of the dorsum and associated demineralization of the anterior and posterior clinoids. Complete destruction of the dorsum of the sella was encountered in only 4 patients and did not necessarily correlate with a particularly active form of the disease (Fig. 1, *A* and *B*). The fact that the disease was chronic in the patients examined in this series may well account for the high percentage of positive findings in the sellar region. Steinbach *et al.*[21] also noted a significantly large number of positive findings of sellar destruction (81 per cent).

The second most common feature was prominence and enlargement of the frontal sinuses; this was present in 23 of 31 patients (Table II). Contrary to the observation of Steinbach *et al.*[21] there was no sig-

TABLE I

OSSEOUS CHANGES OF THE SELLA TURCICA

Changes	No. of Cases
Complete destruction of the dorsum of the sella	4
Generalized enlargement of the sella with rarefaction of the dorsum	19
Slight enlargement of the anteroposterior diameter of the sella	6
No apparent change	2
Total	31

TABLE II

ROENTGEN FINDINGS OF ACROMEGALY

Site and Changes	Appositional growth of bone, thickening and osseous overgrowth	Osseous resorption and bone modeling	No abnormal findings
	No. of Cases	No. of Cases	No. of Cases
Skull (31 Cases)			
Cranial vault—diffuse manifestations	11	4	7
Cranial vault—circumscribed manifestations	7	2	
Occipital spur formation	12		19
Prominence and enlargement of sinuses			
Frontal	23		8
Maxillary	21		10
Mastoid enlargement	13		18
Hands (23 Cases)			
Heads and bases of phalanges	6		17
Phalanges and metacarpal shafts	6	3	14
Phalanges and metacarpal shaft elongations (endochondral ossification)	2		21
Joint cartilage (widening of joint spaces)	14		9
Osteoarthritis	11		12
Exostoses	9		14
Large tufts of terminal phalanges	11		12
Diffuse osteoporosis		7	16
Feet (13 Cases)			
Shafts of phalanges		11	2
Heads and bases of phalanges and metatarsals	10		3
Tufts of distal phalanges	8		5
Tarsals	3	6	4
Osteoarthritis	8		5
Joint cartilage hypertrophy (widening of the joint space)	5		8
Thoracic Spine and Thoracic Cage (20 Cases)			
Length of vertebral bodies (particularly anterior circumference apposition)	10	5	5
Intervertebral disks	12	12*	
Calcified disks	4		16
Osteoarthritis	15		5
Increased anteroposterior diameter of the chest	12		8
Anterior bulging of the sternum	16		4
Lumbar Spine (6 Cases)			
Intervertebral disks	6		
Length of vertebral bodies (modeling of posterior circumference of bodies)		4	2

* Some cases showed both resorption and bone growth at different levels.

nificant distribution of the male and female patients in this respect. Two-thirds of the entire group also showed correspondingly large maxillary antra. An evaluation of the degree of pneumatization of the periantral air cells was quite difficult. However, it was felt that about one-third showed probably increased pneumatization. The mastoid tip in particular often showed extensive pneumatization and prominence. About one-third of all patients, most of them male, showed marked prominence of the occipital

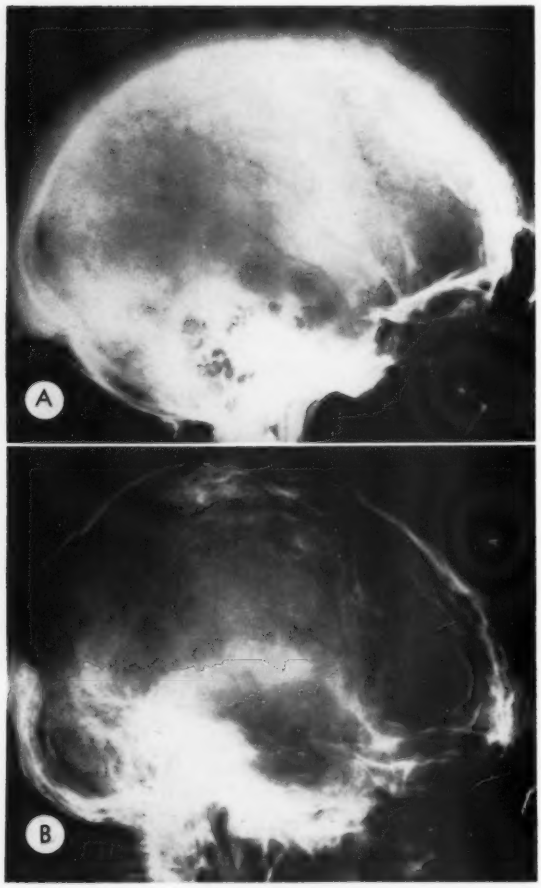

FIG. 1. (*A*) Lateral roentgenogram of the skull showing complete destruction of the dorsum of the sella and posterior clinoids with marked thickening of the entire calvarium and hyperostosis frontalis interna. The occipital protuberance is quite prominent, forming a spur-like hyperostosis at the point of insertion of the nuchal ligament. (*B*) There is almost complete destruction of the dorsum of the sella and the posterior clinoid processes. Note the selective thinning of the parietal bones.

protuberance (Fig. 1*A*). The occipital protuberance projected in the form of a spur apparently formed by marked bone apposition at the point of the insertion of the nuchal ligament.

One of the most interesting observations which has been made in the literature on acromegaly is the presence of both thinning and thickening of the cranial vault.[13,18] This either involves the entire cranial vault or is localized to the frontal and parietal bones. In our series, thickening of the tables appeared to be much more common than thinning. Localized thickening presented most often as hyperostosis frontalis interna. Localized thinning was most commonly seen in the region of the parietal bone (Fig. 1*B*). Both appositional and resorptive changes resulted in the loss of the typical differentiation between the external and internal tables and the diploë. The diploic space exhibited a marked increase in density and a coarsening of its trabecular pattern. A gradual obliteration of the entire diploic space takes place as the disease progresses. There was often associated soft tissue proliferation, probably secondary to thickening of the galea aponeurotica. This proliferation, however, appeared to be independent of osseous hypertrophy. Prominence of the supraorbital ridges and zygomatic arches was a salient feature. The enlargement and elongation of the mandible was difficult to evaluate from our series because most of the roentgenograms failed to show the entire mandible and, hence, did not allow proper interpretation of this region.

HANDS

The first classic description of the so-called "spade hand" dates from 1886.[19] These changes are predominantly caused by soft tissue hypertrophy. Eighteen of 23 patients examined in this series showed marked thickening of the soft tissues of the fingers (Fig. 3). The typical squaring and thickening of the phalanges and metacarpals, however, was only seen in one-fourth of all patients. Three of 23 patients exhibited narrowing of the shafts of the phalanges. This phenomenon was first described by Marie[19] and appears to be limited to juvenile patients. The apparent narrowing is probably caused by a combination of accentuated growth in length produced by excessive endochondral activity and abnormal resorption of bone along the shaft.[7]

The most characteristic changes in the hands are the abnormally wide joint spaces, undoubtedly caused by marked hypertrophy of cartilage (14 of 23 cases.)[3] De-

generative osteoarthritic manifestations were also present and are commonly seen in both younger and older age groups affected with this disease. Small exostoses occur in the areas of tendon or ligament insertion. These changes are best seen in the heads of the metacarpal bones. The enlargement of the ungual tufts of the terminal phalanges is one of the salient features more commonly seen in males. Some patients may show a combination of hypertrophic changes of the distal phalanges associated with osteoporosis, thinning and resorptive processes of the proximal phalanges and metacarpals. This combined type, however, was seen in only 2 patients and appeared to be quite rare.

FEET

The changes of the osseous structures of the feet are similar to those of the hands, combined hypertrophic and resorptive changes being very frequent. Nearly all of our cases showed characteristic narrowing of the shafts of the phalanges contrasting sharply with the disproportionately broad and square bases and heads. At the same time, there was usually thickening and a square appearance to the shafts of the metatarsals (Fig. 4). These changes probably best attest to the dynamics of the disease, showing marked resorptive changes along the shafts of the phalanges as well as apposition and new bone deposition in other areas of the same osseous structures.

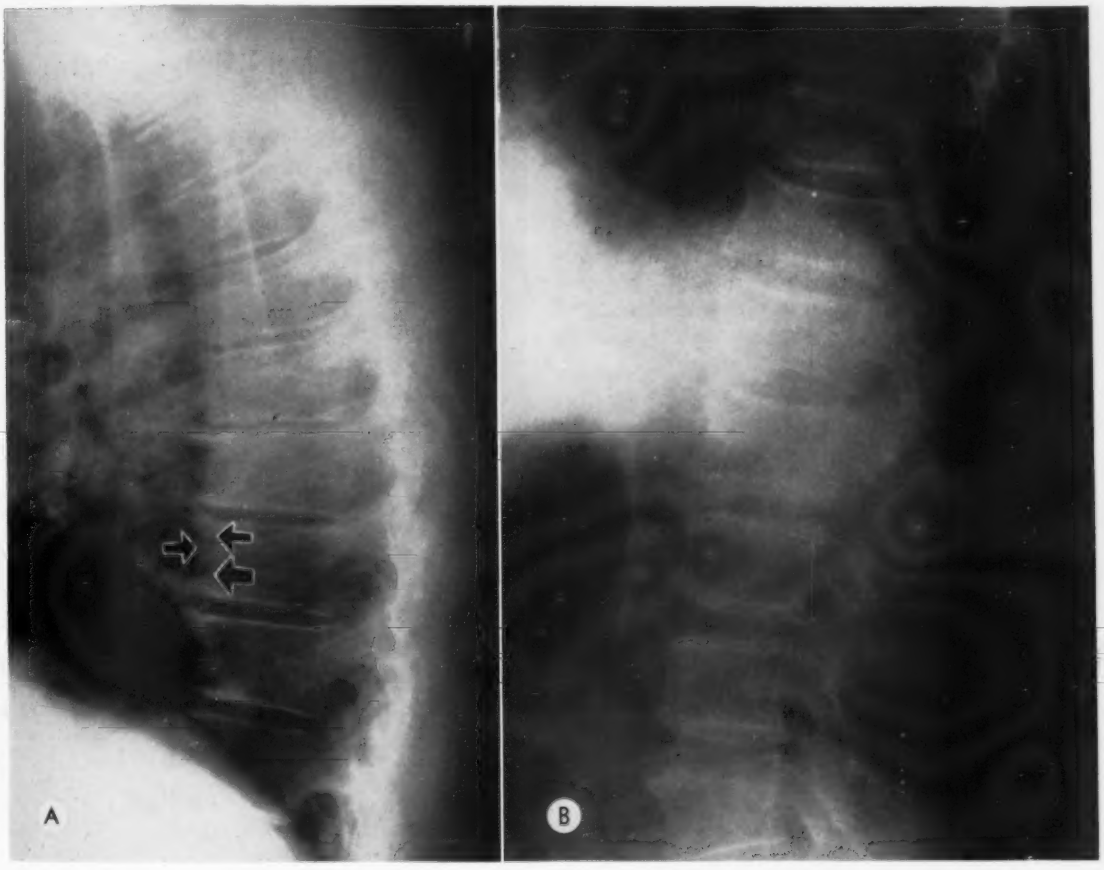

FIG. 2. (*A*) Lateral roentgenogram of the dorsal spine demonstrating the increased length of the dorsal vertebral bodies caused by appositional new bone formation along the anterior aspects. The old anterior margin can still be seen as a faint cortical shadow (arrows). (*B*) Note the marked concavity of the posterior aspect of the lumbar vertebrae caused by bone resorption. There is marked widening of the lumbar disks and also some osteoarthritic changes.

Fig. 3. Marked hypertrophy of the ungual tufts, widening of the metacarpophalangeal joint spaces (secondary to cartilage hypertrophy), and spur and exostosis formation at points of tendon insertion are the salient features. Note the marked thickening of the soft tissues.

In our series, some of the metatarsals also showed thinning, which is considered to be a rather rare finding. Polyarthrosis of both phalanges and metatarsals was commonly demonstrated. Hypertrophy of the joint cartilage was less commonly seen in the lower extremities (5 of 13 cases).

SPINE

The first classic description of the changes of the spine was published by the Austrian pathologist, Erdheim.[10] Since then, many authors have re-emphasized the importance of the accentuation of the physiologic kyphosis of the upper dorsal spine and the compensatory hyperlordosis of the lumbar spine in the diagnosis of this disease.[5,6,7,10,13,18,21,23] The most distinctive changes, however, are undoubtedly those caused by the selective apposition and resorption of bone along the dorsal and lumbar spine.[21] In this series, there appeared to be a significant difference in the changes of the dorsal and lumbar vertebrae. The dorsal vertebrae predominantly showed apposition, particularly along the anterior aspect of the vertebral bodies (Fig. 2*A*). These changes appear to be characteristic of the disease and deserve special attention when reading lateral chest roentgenograms. Conversely, the lumbar spine predominantly showed modeling of the posterior aspects of the vertebral bodies, undoubtedly secondary to resorptive processes (Fig. 2*B*). Both lumbar and dorsal spines may show increased height of the intervertebral disk spaces. In a few instances, however, there was also evidence of degeneration and secondary narrowing of some of the disk interspaces. This was only seen

Fig. 4. Posteroanterior roentgenogram of the foot. Exaggerated overmodeling of the shafts of the phalanges contrasts with the hypertrophic changes of the heads and bases. Septa can be discerned in the soft tissues, indicative of edema.

in the region of the lower dorsal spine. The upper dorsal spine and the lumbar spine showed a uniform increase in the intervertebral disk spaces. Osteoarthritic changes were observed in 15 of 20 cases. The spurs seen in these patients, however, are much more uneven and irregular than those commonly associated with degenerative osteoarthritic disease in older age groups. Furthermore, there appeared to be a significant absence of sclerosis of the end plates of the vertebral bodies, which is characteristic of nonacromegalic degenerative osteoarthritis. These changes are believed to differentiate the osteoarthritis seen in acromegaly from that seen in the old age groups.

THORACIC CAGE

The anteroposterior diameter of the chest is often increased in acromegalic patients; this is due to the more or less horizontal position of the manubrium and the associated ventral displacement of the sternum. The ventral ends of the ribs are often thickened and the costochondral cartilage junction may show knotty proliferations characteristic of the acromegalic rosary. Finlay and MacDonald[13] first commented on the fusiform widening of the costochondral junctions, which was present in 50 per cent of their cases. Waine *et al.*[23] felt that the cartilage of the costochondral junction was surrounded and enveloped in a sheath of new bone formed by the proliferating periosteum. Resorptive processes along the ribs are quite rare and are usually localized to only a few segments.

HIP JOINTS

In addition to the tabulated changes (Table II), one may frequently see degenerative changes of the hip joints. It is postulated that these changes are caused by a qualitatively inferior cartilage which degenerates early and hence exposes the osseous components of the joints to friction. In these areas very severe reactive sclerosis may be encountered and there may in fact be partial destruction of the osseous components. Genuine degenerative osteoarthritic spur formation is then seen.

DISCUSSION

The diagnosis of acromegaly remains primarily a clinical problem. Roentgenologic studies, however, offer a method for objective comparison of the progression or regression of changes caused by this disease. One of the aims of careful follow-up is to determine whether the disease is quiescent or active and what effect therapy has had.

Three of our patients have been followed for a number of years with multiple roentgenographic studies both prior to and after definitive therapy. These patients showed progressive osseous and soft tissue changes before therapy. The regression of these changes after radiation therapy or surgical extirpation of the tumor was not always dramatic. Initially there was a marked reduction of the soft tissue swelling of the fingers. This observation has been confirmed by many authors but attempts to quantitatively assess this finding have thus far failed.[8,15,17] The regression of soft tissue swelling was only observed in the first few weeks and failed to reach complete restoration to normal, thus leaving residual enlargement which was probably due to irreversible connective tissue hyperplasia. Neurologic and ophthalmologic defects also showed excellent early regression, since these changes had been caused by pressure of the tumor mass which responded rapidly to therapy. Changes of the osseous system appeared to show little effect from the changed hormonal levels. In fact, with the exception of recalcification of the clinoids and sella, there was hardly any regression of the existing osseous changes. Only 1 patient showed definite bone apposition along the shafts of the tarsal phalanges which had demonstrated the classic modeling and thinning during the active form of the disease. These changes became apparent one and one-half years after a tumor dose of

3,500 r had been delivered via five external ports to the sellar region.

Pantopaque ventriculography, exactly outlining the floor of the third ventricle, will aid greatly in the evaluation of the response of suprasellar tumor masses treated with external radiation therapy or implantation of radioactive materials.

SUMMARY

Bone apposition along the anterior aspect of the vertebral bodies of the dorsal spine and the increased height of the intervertebral disks are considered to be characteristic roentgenologic features of acromegaly. The absence of sclerosis of the end plates of the vertebral bodies offers a criterion for differential diagnosis between degenerative and acromegalic osteoarthritis of the spine. A combination of bone apposition and resorption, characteristic of this disease, is particularly frequent in the foot.

The high incidence of sellar changes is felt to reflect the long mean duration of the disease in this series.

Soft tissue swelling as well as neurologic and ophthalmologic changes caused by pressure of the tumor mass show a marked and early response to treatment. The osseous changes are, in general, irreversible.

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PRIMARY HYPERPARATHYROIDISM: A CORRELATION OF ROENTGEN, CLINICAL, AND PATHOLOGIC FEATURES*

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THAT the parathyroid glands are involved in the regulation of the calcium stores of the body and in maintenance of the mineralization of the skeleton has been known for many years.[1,16,18,19,24] The roentgen appearance of grossly abnormal bone produced by excessive secretion of the parathyroid hormone has also been adequately described.[6,21,22] The problem of determining the earliest roentgen manifestations of hyperparathyroidism, however, has not been extensively investigated, and this study will attempt to do so.

PHYSIOLOGY

Phosphate homeostasis is maintained by the action of the parathyroid hormone on the renal tubular transport of phosphate. The parathyroid hormone also maintains the blood calcium level by mobilization of calcium from bone mineral. An excess of the hormone, therefore, causes hypercalcemia and hyperphosphaturia.[7,12] If phosphate loss is not replaced by dietary phosphate intake, hypophosphatemia results.

Evidences of excessive parathyroid activity result either from primary parathyroid overfunction, such as in adenoma or primary hyperplasia, or from secondary hyperfunction as in steatorrhea or renal failure. The initiating event for secondary hyperparathyroidism is probably hypocalcemia; whether hyperphosphatemia has a similar effect is controversial. The kidney is also affected by hyperparathyroidism. Initially, functional impairment consisting of inability to concentrate urine and to conserve phosphate is seen. Later, calcium salts are found in the cells and lumen of the convoluted tubules. Still later, calcification and fibrosis of the parenchyma progress to renal insufficiency.[15] Secondary pyelonephritis and parenchymal calcification further complicate the roentgenologic appearance of calcium deposition.

Much has appeared in the literature on metastatic calcification of the soft tissues. This calcification is of two types: (1) that due to hypercalcemia, as in calcific conjunctivitis, and (2) that resulting from a combination of hypercalcemia and the hyperphosphatemia of renal failure, as with keratopathy and calcification of the alveoli of the lungs, vessel walls, gastric mucosa, cartilaginous and periarticular structures, and other tissues. Curiously, metastatic calcification of soft tissues occurs concomitantly with decalcification of the skeleton.

Hyperparathyroidism may cause disturbances of the urinary tract and skeletal system; it may give rise to symptoms of resultant acute hypercalcemia, or it may be present without evident symptoms. The urinary tract symptoms consist of hyposthenuria, polyuria, nocturia, polydipsia, and dehydration, most commonly observed in patients with severe hypercalcemia. The most common urinary tract complications are nephrolithiasis, pyelonephritis, and nephrocalcinosis. Skeletal involvement may first be manifested by fractures with little or no trauma, but rarely by bone pain and tenderness. Localized expansion, bending,

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TABLE I

GROSS PATHOLOGY IN 112 CASES OF PROVED HYPERPARATHYROIDISM*

Single adenoma	92
Multiple adenomas	10
Primary (wasserhelle) hyperplasia	9
Carcinoma with metastasis	1

* From: Black, B. M.[4] Hyperparathyroidism. Charles C Thomas, Springfield, 1953, p. 20.

or fracture may cause bone deformities.

The clinical symptoms of acute hypercalcemia are anorexia, nausea, vomiting, constipation, abdominal distention, thirst, nocturia, polyuria, weakness, and, when the serum calcium is exceedingly high, lethargy, somnolence, or psychosis. Peptic ulcer is extremely common in patients with hyperparathyroidism. Hellström[15] reported that 33 per cent of the men and 5 per cent of the women in his series had peptic ulcers. Many more have ulcer-like symptoms, approximately 45 per cent of all patients with hyperparathyroidism presenting themselves because of gastrointestinal disturbances.

The keystone of diagnosis is hypercalcemia, often of mild degree, requiring unusually accurate laboratory procedures for recognition and evaluation. Hypophosphatemia, formerly the rule, is now found in only about 40 per cent of cases. Hyperphosphaturia, demonstrated by a low rate of tubular reabsorption of phosphate, is uniformly present. Similar chemical abnormalities occur in other disorders, such as myeloma, sarcoidosis, and neoplasms.

PATHOLOGY

Primary hyperparathyroidism may be caused by a single adenoma, multiple adenomas, primary hypertrophy and hyperplasia, chief cell hyperplasia, or carcinoma.[4,8] The incidence at the Mayo Clinic of these lesions as causes of hyperparathyroidism is shown in Table 1. Chief cell hyperplasia may be associated with islet cell adenomas of the pancreas, chromophobe adenomas of the pituitary, and adrenal cortical hyperplasia.[8]

Osteitis fibrosa cystica generalisata affects the entire skeleton. The earliest and most severe lesions appear in regions where bone production and the invariably associated bone destruction or remodelling occur most rapidly.[16] In children, some of the most pronounced changes are in the metaphyses adjoining the growth plate where calcified cartilage is replaced by primitive bone which, in turn, undergoes remodelling through the mechanism of osteoclastic resorption and surface accretion[16] (Fig. 1). In both adult and child, early lesions usually appear along the periosteal surface of the cortex, particularly at the point of tendinous or ligamentous attachments. Sites of injury, mechanical pressure, or tension, with osteoclastic bone destruction and osteoblastic repair, appear to be more sensitive to the parathyroid hormone. The reaction is the opposite of that in acro-

FIG. 1. Hyperparathyroidism resulting from hyperplasia in a ten week old child. A diffuse demineralization and bending deformities of the distal ends of the femora, tibiae, and fibulae are seen. The most marked bone destruction is in the distal femur where a horizontal linear radiolucent shadow is visible proximal to the zone of provisional calcification. (Courtesy, Doctor G. Melvin Stevens, Palo Alto Medical Clinic.)

TABLE II

AGE AND SEX OF PATIENTS WITH BONE INVOLVEMENT STUDIED ROENTGENOGRAPHICALLY

	No. of Cases	Males	Females	Average Age (yr.)
Total number of patients	47	24	23	45 (Range 17–70)
Patients with bone involvement	13 (28%)	7	6	42 (Range 17–64)

megaly in which bone production is greatest at points of stress. Weinmann and Sicher[24] found characteristic histologic lesions of osteitis fibrosa cystica in bone after the extraction of an infected tooth, and adjacent to inflammatory polyps in the maxillary sinus of patients with moderate renal insufficiency and secondary hyperparathyroidism.

The mechanism by which the parathyroid hormone causes bone to be reabsorbed is not fully understood, but probably the resorption is effected by osteoclasts altering the bony matrix and minerals which become soluble and pass into the tissue fluids. This dissolution of bone substance involves the stable, fully mineralized bone, most of which is not accessible to the rapidly acting transfer mechanism. The bone reabsorbs only along the surfaces of the finer structures.[18,19,24]

The well-developed histologic lesion is caused by osteoclastic resorption of bone, associated with reparative fibrosis and formation of poorly calcified immature new bone. The compact, cortical bone is replaced by spongy bone. Localized accumulations of fibrous tissue and giant cells, called "brown tumors," may produce expansion of bone similar to giant cell and other benign tumors. "Brown tumors" undergoing necrosis and liquefaction produce cysts.[16]

ROENTGEN FINDINGS

Forty-seven patients (24 males and 23 females) from a group of 61 were selected for study because of the availability of their roentgenograms. The average age of patients with roentgenographic lesions was three years less than for the group as a whole (Table II).

Originally, the diagnosis of hyperparathyroidism was made only when the presence of generalized osteitis fibrosa was determined roentgenologically, or by bone biopsy of advanced lesions. Following the report of Albright *et al.*,[3] the importance of renal complications was emphasized in the early diagnosis of the disease. Refinements in laboratory methods have further decreased the incidence of skeletal lesions detected by ordinary roentgenographic methods. The reported incidence of bone lesions has varied considerably; the general consensus of opinion ranges from about one-third to 40 per cent[4,5,15,22] In most reports, the extent of skeletal surveys was not described.

In the group reported here, 13 of 47 patients (28 per cent) had bone lesions detected roentgenologically (Table III). Considering only extensive skeletal surveys that included roentgenograms of the hands, long bones, skull, and pelvis, 13 patients of a total of 28 (46 per cent) showed bone

TABLE III

INCIDENCE OF BONE INVOLVEMENT

	No. of Cases	Bone Involvement
Total number of patients examined	47	13 (28%)
Patients with complete skeletal roentgen study	28	13 (46%)
Patients with roentgen study of hands	36	13 (36%)

Fig. 2. (*A*) Advanced bone lesions with cortical erosion, most obvious along the lateral (thumb) aspect of the middle phalanges. The shafts of the middle phalanges have become narrow. Destructive lesions of the outer cortical surface are also visible in the proximal phalanges, metacarpals, and the distal lateral surface of the radius and medial surface of the ulna. The cortex of the sesamoid bones is rough. Extensive destruction of the shafts of the distal phalanges and of the terminal tufts has produced shortening of the terminal segments of the fingers, clinically resembling "clubbing." A cyst-like destructive lesion is present in the head of the fifth metacarpal. Irregular decalcification exists. (*B*) The same hand following parathyroidectomy. Despite healing, bone texture is not normal. Bones previously involved by destruction are now more dense than normal. The large destructive lesion in the fifth metacarpal head has not healed, indicative of a cyst.

lesions. In our experience, all patients with roentgenographic evidence of generalized osteitis fibrosa had characteristic lesions in the middle phalanges of the hands. Of the 36 patients who had roentgenograms of the hands, the incidence of 36 per cent probably represents the true incidence of bone lesions in the entire series (Table III). Of the 41 patients examined since 1954, 10 showed roentgenographic evidence of bone lesions (24 per cent).

In the classic case of hyperparathyroidism, roentgenograms show erosion of the cortical surfaces of bones, demineralization of bones, localized destructive lesions, and calcification of the soft tissues.[6,22]

Cortical erosion. In advanced cases of hyperparathyroidism, all of the cortical surfaces may be involved. In certain sites of predilection, however, cortical erosion may be visible before other manifestations of the disease. The sites most frequently involved are the lateral (radial) aspect of the middle phalanges of the hands and the tufts of the distal phalanges (Fig. 2*A*). In patients with minimal involvement, erosion is seen only near the phalangeal base in the transitional zone between the wide epiphyseal end and the slender shaft (Fig. 3). Other regions where early subperiosteal cortical erosion may be present are the articulations of the distal end of the clavicle (Fig. 4), the symphysis pubis, and the sacroiliac joints. Similar erosions may be

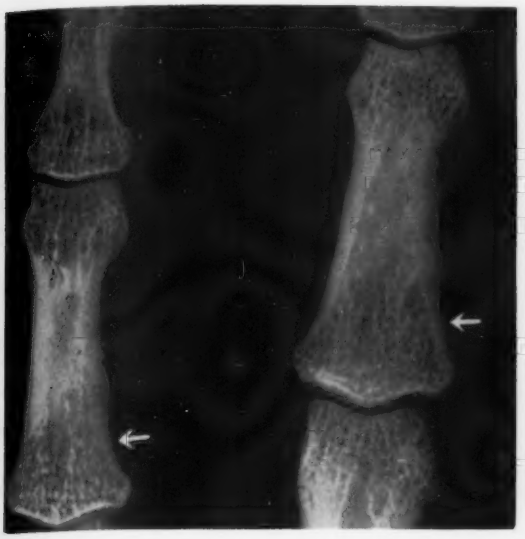

Fig. 3. Localized erosion involving the lateral cortical surfaces of the third and fourth middle phalangeal bases (arrows). The bones do not otherwise appear abnormal.

noted in the lamina dura (the cortical bone surrounding the root of the tooth), the calcaneal tendon insertions at the plantar or Achilles surfaces (Fig. 5), the medial aspect of the proximal end of the tibia (Fig. 6), the ischial tuberosities (Fig. 7*A*), the greater and lesser trochanters, and the crests of the ilia. Cortical erosion of the middle phalanges was present in all 13 patients with bone disease (Table IV).

The subperiosteal cortical erosion has the same roentgenologic appearance in different bones. The normal, smooth outer

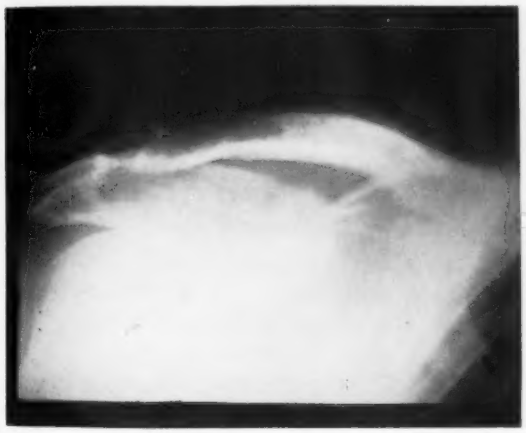

Fig. 4. Erosion of the distal end of the clavicle and adjacent surface of the acromion process.

Fig. 5. Erosion of the calcaneus at the attachment of the plantar fascia and, to a lesser extent, at the Achilles tendon (arrows). These lesions, along with those of the clavicle, sacroiliac joints, symphysis pubis, and ischial tuberosities, resemble the lesions of these same structures seen in ankylosing spondylitis, rheumatoid arthritis, and Reiter's disease.

Fig. 6. A localized defect (arrow) in the upper medial cortex of the tibia is a common manifestation of hyperparathyroidism. The lesion may involve a longer segment of the tibia, but is always shallow. It corresponds in position to the attachment of the collateral tibial ligament.

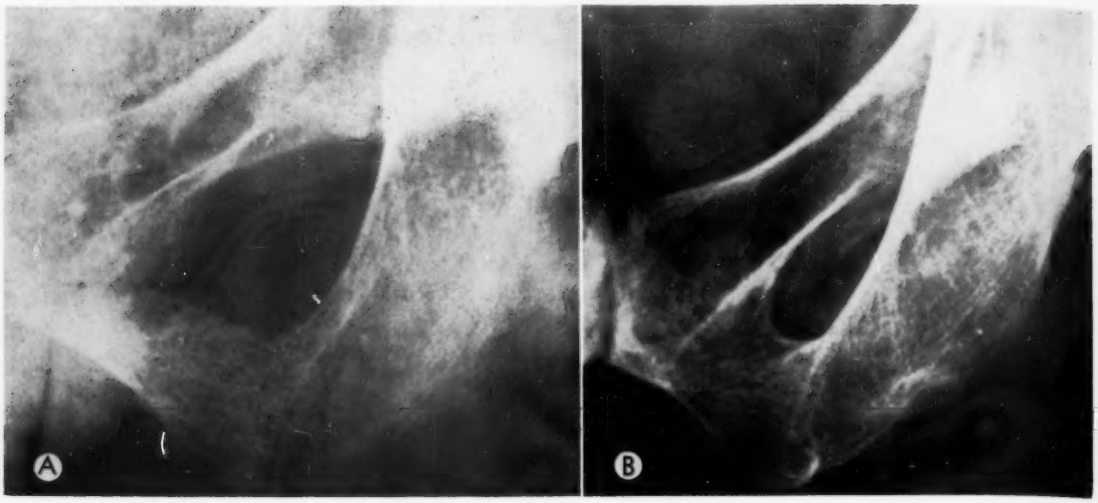

Fig. 7. (*A*) The lateral inferior cortex of the ischium and ischial tuberosity has a loss of its compact cortical surface. (*B*) Eight years after parathyroidectomy, the cortex has been restored.

surface of the cortex at first loses its sharp demarcation from the surrounding soft tissues. Later, areas of destruction alternating with normal appearing bone may produce spicules perpendicular to the cortical surface. In tubular bones, these changes are associated with vertical striations of the middle and deeper layers of the cortex caused by a widening of the haversian canals, the longitudinal spaces enclosing the blood vessels and surrounding connective tissue (Fig. 8*A*). Such typical subperiosteal resorption occurs in no other disease; when demonstrated in cases of osteomalacia due to gastrointestinal malabsorption or renal tubular dysfunction, or in cases of renal glomerular failure or pseudohypoparathyroidism, it is the result

Table IV

SITES OF BONE INVOLVEMENT

Age	Sex	Cortical Erosion of Phalanges of Hands	Absence of Lamina Dura	Cysts	Skull Mottling	Demineralization
49	F	+		○	+	+
36	F	+	+	+	+	+
41	F	+	+	+	+	+
17	F	+	+	+	○	+
64	F	+		+	○	+
36	F	+	○	○	○	○
56	M	+	○	+*	○	○
29	M	+	○	+	+	○
64	M	+	○	+†	○	+
23	M	+	○	+*	+	○
51	M	+	Upper + Lower ○	+*		+
44	M	+		+	+	+
41	M	+		+		+

* Mandible.
† Maxilla.

of secondary hyperparathyroidism.[9,17,23] Lesions resembling those of hyperparathyroidism may involve the calcaneus, sacroiliac joints, symphysis pubis, ischial tuberosity, and lateral clavicle in cases of ankylosing spondylitis, rheumatoid arthritis and Reiter's disease, but in these diseases the phalanges are not affected.

Demineralization of the skeleton. This is not always visible on roentgenograms, even in the presence of other evidence of bone involvement. Of the 13 patients in this series with bone involvement, the bones of 9 showed diminished density (Table IV). The demineralization usually differs from that of osteoporosis and osteomalacia in that it is not homogeneous. In hyperparathyroidism, bone of diminished density alternates with bone of apparently normal density, producing a mottled or striated appearance. This may be most obvious in the calvarium (Fig. 9*A*) but is also visible in other bones. Ultimately, when demineralization is extensive, it may appear homogeneous.[11] In the present series there were 10 patients with generalized homogeneous decalcification without evidence of subperiosteal resorption or destructive lesions (Fig. 10). All were middle-aged or elderly, and 8 were women. Following parathyroidectomy, roentgenograms showed that in these patients demineralization remained, and was

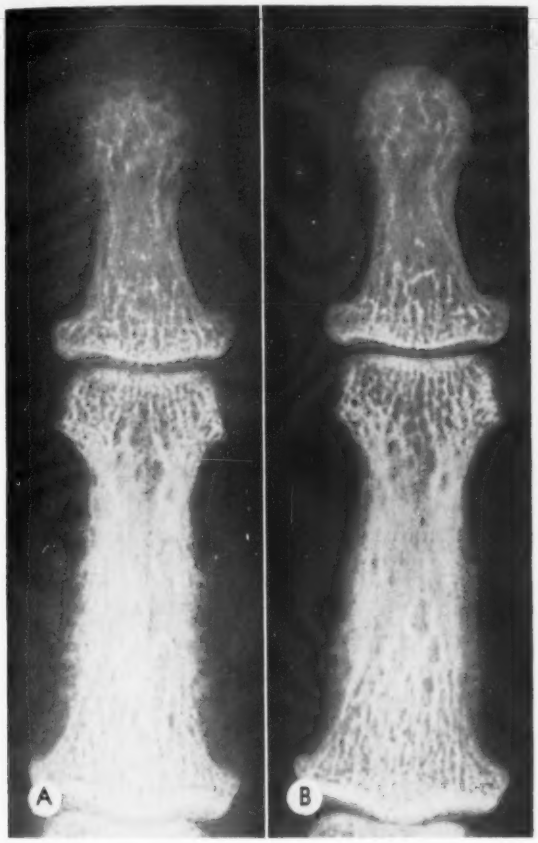

FIG. 8. (*A*) Extensive cortical destruction of the middle phalanx with horizontal spiculation. Vertical striations are prominent due to erosion of the walls of the haversian canals. Some bone resorption of the distal phalangeal tuft is visible. (*B*) Three months after parathyroidectomy, the periosteum has deposited a continuous sheet of bone, filling in the spaces between the horizontal spicules.

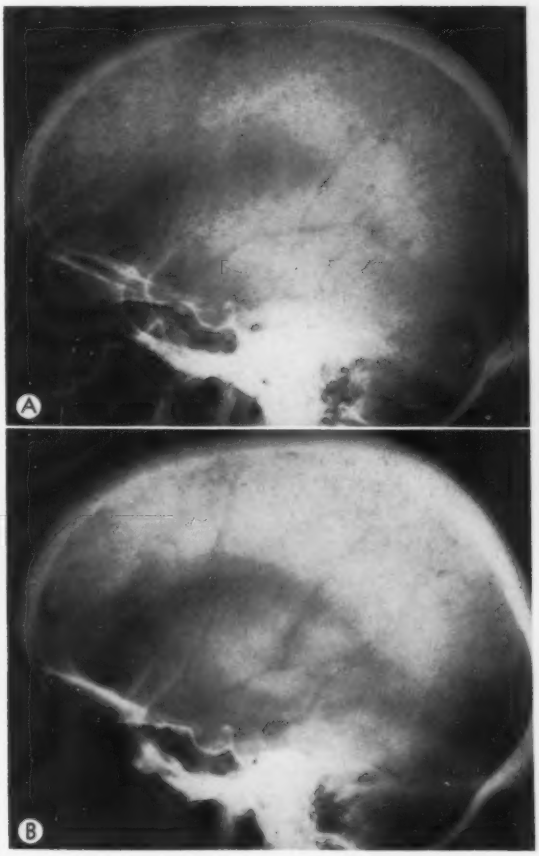

FIG. 9. (*A*) The calvarium has a mottled appearance along its periphery, most obvious distal to the inferior temporal line. The grooves for the peripheral branches of the meningeal vessels are less obvious than in *B*. (*B*) Seven years following parathyroidectomy, the calvarium is more dense.

Fig. 10. In a sixty-eight year old female with diffuse homogeneous demineralization of all bones, the cortex is thin but sharply defined. In roentgenograms after parathyroidectomy, no change in mineralization of the skeleton was apparent, suggesting that the visible bone disease was osteoporosis rather than osteitis fibrosa.

Fig. 11. "Brown tumor" focus in the right side of the mandible. The lesion has extended beyond the normal periosteal margin of the mandible and is enclosed in a thin layer of periosteal bone. The lamina dura, as seen in dental roentgenograms, was intact.

probably the result of postmenopausal or senile osteoporosis. Pugh[21] also noted diffuse demineralization without evidence of osteitis fibrosa in some patients and concluded that they may have had osteoporosis. Others have found demineralization of the skeleton, presumably from hyperparathyroidism, in the absence of other evidence of hyperparathyroid bone disease.[11,15]

Localized destructive lesions. Also known as "brown tumors," these are frequently found in the jaws or facial bones, but may be present in any part of the skeleton. In the shaft of a long bone they may be eccentric. Roentgenographically, they are usually sharply demarcated and may expand the cortex (Fig. 11). A single lesion of one bone, or many lesions involving several bones, may be present. In 11 patients with destructive lesions there was subperiosteal resorption of the middle phalanges of the hands, but the surrounding bone did not appear demineralized in 3 (Table IV). The "brown tumors" heal after removal of the parathyroid tumor, or after local radiation therapy or curettage. Some undergo cystic degeneration, persisting as defects in the bone after the primary disease has been cured. The cystic lesions do not differ roentgenographically from the tumors and can be recognized only by their failure to heal (Fig. 2*B*). All 4 patients in this series with localized destructive lesions of the jaw had persistence of the lamina dura (Table IV). In some cases the tumor encroached upon the adjacent lamina dura, producing a localized destruction but with the lamina dura of the other teeth remaining intact (Fig. 12). One patient had a tumor of the mandible with absence of the lamina dura but with normal lamina dura in the maxilla.

The degree of bone involvement compared with blood chemical determinations. This is shown in Table V. Serum calcium and phosphorus determinations were obtained in all 13 patients. No correlation could be found between the values of these chemical determinations and the presence or absence of bone involvement. The serum alkaline

Fig. 12. Destructive lesion ("brown tumor") involving the cancellous bone about the roots of the right lower cuspid and first bicuspid. The lamina dura is destroyed in the region of the tumor but is intact elsewhere.

phosphatase, however, was a reliable guide to the presence or absence of osteitis fibrosa and an index to the extent of the bone disease. The alkaline phosphatase in patients without roentgenologic evidence of bone lesions ranged from 3.0 to 7.7 Bodansky units and averaged 5.4. Two patients with values of 9.0 and 25.5 Bodansky units were eliminated because of the presence of Paget's disease, which also produces an elevation in the alkaline phosphatase. In patients with roentgenologic evidence of

Table V

Degree of bone involvement in relation to blood chemical determinations

Age	Sex	Bone Involvement	Alkaline Phosphatase (Bodansky Units)	Calcium (mg. per 100 cc.)	Phosphorus (mg. per 100 cc.)
64	M	Minimal	5.0	14.4	2.4
36	F		6.2	14.2	3.0
41	M		11.0	16.4	2.3
49	F	Moderate	8.0	15.4	3.5
51	M		11.0	14.5	1.1
64	F	Severe	11.0	12.2	1.8
23	M		17.7	16.0	1.7
17	F		33.2	17.6	2.6
51	M		35.0	12.0	1.4
44	M		36.5	14.5	2.7
41	F		46.0	13.9	2.4
29	M		56.0	15.4	3.0
36	F			15.6	2.2

Fig. 13. A roentgenogram several years after parathyroidectomy demonstrates healed bone lesions. Bone bridges the symphysis pubis and sacroiliac joints. Bone extends from the ischial tuberosities and lesser trochanter of the right femur into the adjacent soft tissues. On preoperative roentgenograms, destruction of cortical bone was visible at these sites.

bone disease, the range was from 5.0 to 56.0 Bodansky units and the average was 23.1. These patients were divided into three groups, depending on the extent of bone disease. The serum alkaline phosphatase varied directly with the severity of the bone disease. Only 1 patient had an alkaline phosphatase determination approaching the normal value (normal 2.0 to 4.5 Bodansky units). A similar correlation has been noted by others.[21]

Healing of bone lesions. In one patient, roentgenographic studies repeated for four years showed slight progression of the bone lesions. Although we have not seen any cases of spontaneous regression, clinically intermittent hyperparathyroidism has been reported.[25] Rapid healing follows parathyroidectomy, with improvement noted on roentgenograms within four to six weeks. The recalcification begins at the periphery of the lesion. In areas of cortical resorption, calcified bone first becomes visible as a thin dense linear shadow parallel to the cortex but separated from it by the intervening fibrous and osteoid tissue. Subsequently, this radiolucent tissue becomes converted to dense bone (Fig. 2*B*, 7*B*, 8*B*, and 9*B*). The localized destructive lesions also begin repair at the periphery with ultimate replacement by dense bone. The mottled decalcified areas become denser than in normal bone, frequently producing exaggerated striations. The newly formed bone may extend along tendons or ligaments, which may result in fusion of the symphysis pubis and sacroiliac joints (Fig. 13). In our experience, the bones usually do not revert to normal but continue to harbor the dense scars or radiolucent cysts. That this bone is susceptible to trauma is demonstrated by 1 of our patients whose jaw was fractured during a dental extraction ten years after parathyroidectomy.

Localized roentgen therapy to the bone lesion also induces healing with the same pattern as that which follows parathyroidectomy.

Kidney lesions. Roentgenologic evidence of urinary calculi was present at one time or another during the course of the disease in 36 (77 per cent) of the patients (Table VI). Defining nephrocalcinosis as diffuse calcification of the renal parenchyma, 4 patients fitted into this category, whereas 32 had discrete renal calculi. The renal calculi varied in size and density, were unilateral or bilateral, and single or multiple. Hellström,[11] defining nephrocalcinosis as multiple small calcified areas in the renal parenchyma, either localized or diffuse, found nephrocalcinosis in 37 of a total of 56 patients. It is known from renal biopsy or autopsy that nephrocalcinosis is present in many patients in whom it could not be detected roentgenographically. Many patients reported having or passing calculi for several years before the diagnosis of hyperparathyroidism was established. All but 1 of the 13 patients with bone disease had renal calculi during the course of the illness. Four of the patients had calculi prior to the onset of bone lesions but not roentgenographically evident at the time bone lesions appeared.

Soft tissue calcification. Except for calcification of the kidneys or conjunctiva, this is not a common feature of the disease. It is present only in severe cases with extensive renal damage. Considerable vascular calcification was noted in 2 patients who had bone lesions and uremia. One patient, without bone or renal disease, had calcifications of the articular cartilages of one wrist which were probably not related to the disease. Another patient, sixty-seven years old, with renal calculi but no bone lesions, had extensive vascular calcification, perhaps the result of arteriosclerosis.

The weight of the parathyroid tumor related to the presence of bone or kidney lesions. Involved parathyroid glands in patients with bone disease averaged more than five times the weight of those in cases without bone involvement (Table VII).

A similar relationship was noted by Woolner *et al.*[26] and Hellström.[13] Hellström reported that the size of the parathyroid neoplasm was not related to the duration of symptoms. In patients with renal disease, he found that small adenomas with a large number of "wasserhelle" cells predominated; in those with skeletal lesions, large adenomas with few "wasserhelle" cells were the rule.

Esophageal and tracheal displacement. Thirty patients had roentgenograms of the pharynx and upper esophagus, 3 showing displacement of the esophagus and 3 of the trachea by an adjacent soft tissue mass. Four of these 6 patients, however, also had thyroid nodules, one definitely causing the tracheal displacement. In the entire series,

TABLE VI

INCIDENCE OF RENAL CALCIFICATION

	No. of Cases	Male	Female
Total number of patients with renal calcification	36 (77%)	20	16
Patients with nephrocalcinosis	4	2	2
Patients with renal calculi	32	18	14

TABLE VII

WEIGHT OF INVOLVED PARATHYROID GLANDS

	Range (gm.)	Average (gm.)
With bone disease	1.0 –11.8	5.02
Without bone disease	0.074– 5.0	0.94

13 patients (28 per cent) had thyroid neoplasms. Four of these were carcinomas and 9 were thyroid adenomas or involutional goiters. The high incidence of coexisting neoplastic changes in the thyroid and parathyroid glands has been reported by others.[13] Wyman and Robbins[27] detected parathyroid adenomas in 20 or 30 patients who were given adequate examinations. However, 23 patients were excluded from their series because the examination had been inadequate or because roentgenograms were not available. They also disregarded the possibility that associated thyroid neoplasms caused the esophageal or tracheal displacement. The lesions most likely to produce displacement of adjacent structures are those of large size, usually easily identified by the surgeon. Despite inherent pitfalls in the interpretation of roentgenograms of the neck and upper mediastinum for location of parathyroid masses, roentgenograms are sometimes of considerable value.

DISCUSSION

The most important roentgenograms for the diagnosis of generalized osteitis fibrosa are those of the hands in the posteroanterior view. In the present series, all patients with roentgenographically demonstrable bone lesions had subperiosteal erosion of the lateral (radial) aspect of some of the phalanges and almost all had erosion of the cortex of the distal phalangeal tufts of one or more fingers. In some cases, not all of the middle phalanges were involved, and, occasionally, the lesions were more obvious in one hand than in the other. Subperiosteal resorption is pathognomonic of generalized osteitis fibrosa, whether it is the

result of primary or secondary hyperparathyroidism. Cases of osteomalacia with mottling of the skull, absence of the lamina dura, subperiosteal resorption, and cyst-like destructive lesions have been described, but these cortical changes are the result of secondary hyperparathyroidism.[9,23]

A review of the literature reveals an overwhelming emphasis on the role of the disappearance of the lamina dura (the cortical bone lining the tooth socket) in the diagnosis of hyperparathyroid disease. Two other lesions of the jaw bones, demineralization and pseudocysts (giant cell osteoclastomas), have been described. It has frequently been reported that one or more of these changes occur in the mandible and maxilla in about half the patients with this disease. Unfortunately, the reported series were small and not well substantiated, with the evidence apparently derived from selected patients. Furthermore, many of the dental changes were based on lateral jaw and skull roentgenograms, which are of little or no value in interpreting the fine trabecular and lamina dura changes.

Of the 13 patients with roentgenologic evidence of bone lesions, 9 dentulous patients had adequate dental roentgenographic examinations. Only 4 of these had absence of the lamina dura. In a recent series of 29 consecutive dentulous patients with histologically proved parathyroid adenomas (including some from this investigation and others which were seen more recently) 2 had, in addition to other skeletal lesions, destruction of the mandible and maxilla. One of these patients had partial loss of the lamina dura about two teeth (Fig. 12). None of the other 27 patients showed abnormal bone changes in dental roentgenograms, although 10 had roentgenographic evidence of disease in other bones.

These observations suggest that, when applied as a sensitive reflection of parathyroid status, jaw bone changes in dental roentgenograms have been overemphasized.

Erosion of the endosteal, cortical surface of bones probably is as advanced, or more so, than that on the periosteal surface and may be observed on some roentgenograms.[16] Its detection is more difficult because of the overlying bone projected upon the roentgenograms and because the normal endosteal bone is less compact and the contour is irregular.

Secondary hyperparathyroidism is an interesting complication following surgical removal of a functioning parathyroid adenoma. The roentgenologic manifestations have not been previously reported, probably because routine postoperative roentgenograms of the hands at frequent intervals are not ordinarily obtained. In 1 of our patients, it was interesting to compare a preoperative roentgenogram (Fig. 14*A*) with a postoperative roentgenogram (Fig. 14*B*) made four months following the surgical procedure. The considerable healing which was seen coincided with a depressed serum calcium (from 12.5 mg. per 100 cc. to 8.5 mg. per 100 cc.) and no appreciable change in the urinary phosphorus (3.1 to 3.4 mg. per 100 cc.). Two and one-half months later (Fig. 14*C*), the destructive bone lesions were more advanced, associated with a drop in the serum calcium to 7.8 mg. per 100 cc. Subsequently, the serum calcium rose to normal values and the bone lesions demonstrated a continuing improvement. The patient had been maintained on a low calcium diet during the pre- and postoperative course because of nephrocalcinosis and moderately impaired renal function. The course of the renal disease did not change following parathyroidectomy.

A problem of concern is that of the frequent disparity in involvement of kidneys and bones in some patients. Albright and Reifenstein[2] expressed the opinion that high calcium diets favor renal disease and prevent bone disease. To substantiate this theory, they demonstrated healing of bone lesions by increased calcium in the diet. Dent[10] observed the same improvement after vitamin D treatment, which is in agreement with our experience.

In the present investigation, the initial

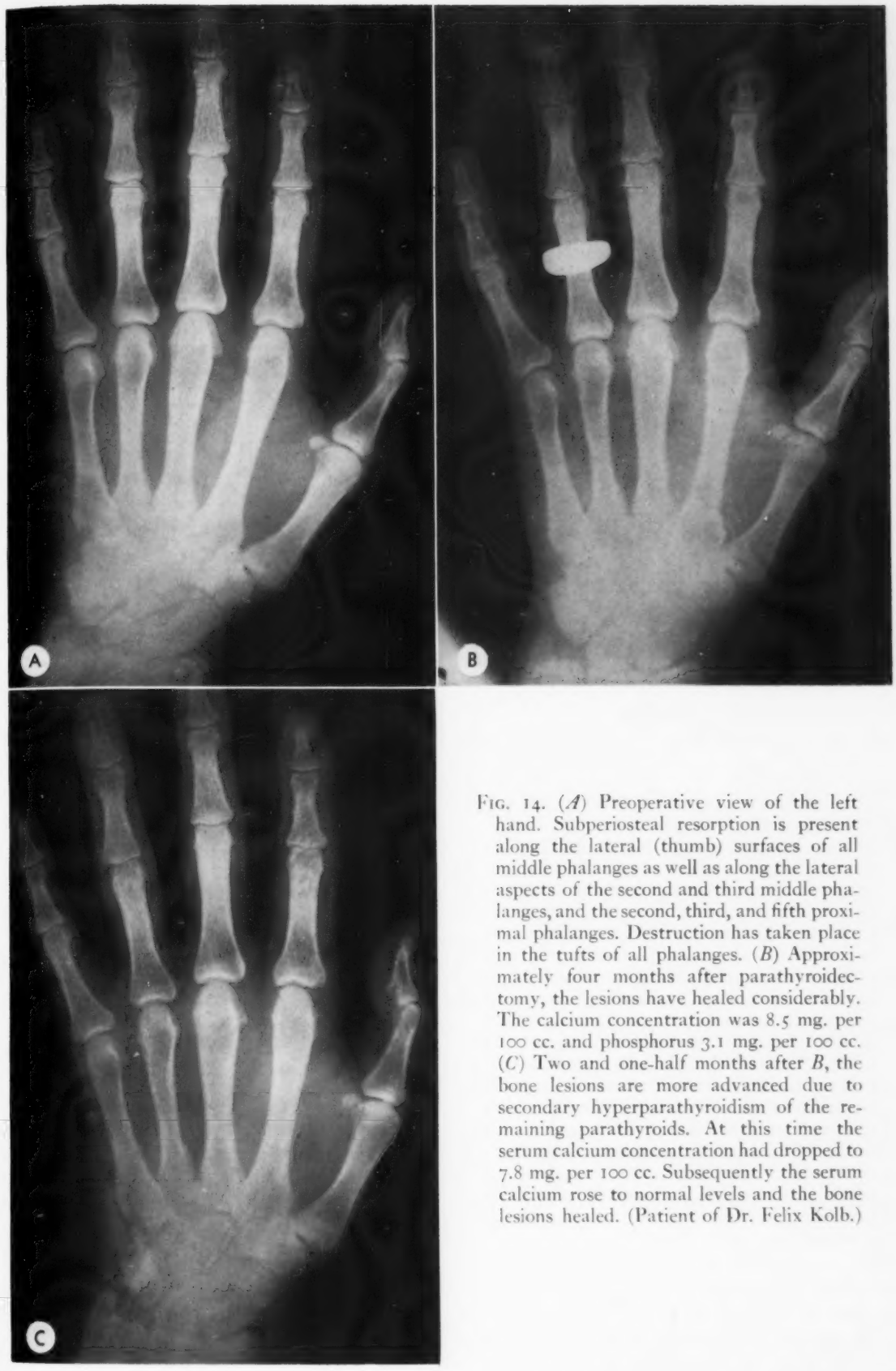

FIG. 14. (*A*) Preoperative view of the left hand. Subperiosteal resorption is present along the lateral (thumb) surfaces of all middle phalanges as well as along the lateral aspects of the second and third middle phalanges, and the second, third, and fifth proximal phalanges. Destruction has taken place in the tufts of all phalanges. (*B*) Approximately four months after parathyroidectomy, the lesions have healed considerably. The calcium concentration was 8.5 mg. per 100 cc. and phosphorus 3.1 mg. per 100 cc. (*C*) Two and one-half months after *B*, the bone lesions are more advanced due to secondary hyperparathyroidism of the remaining parathyroids. At this time the serum calcium concentration had dropped to 7.8 mg. per 100 cc. Subsequently the serum calcium rose to normal levels and the bone lesions healed. (Patient of Dr. Felix Kolb.)

manifestations of hyperparathyroidism in 4 patients were renal calculi without bone disease. Subsequently, the stones were passed and bone lesions developed. The stones did not re-form, even with the continued severe hyperparathyroidism. Retrospective analysis of the diets is apt to be misleading. Following the kidney lesions the patients may have adopted low calcium diets, shifting the burden from the kidneys to the bones.

However, the lack of bone lesions, despite a low mineral diet upon which most of our patients were maintained during their management and metabolic investigations, indicates that diet is not of great importance. Although some patients with severe renal disease and primary or secondary hyperparathyroidism show elevated serum phosphorus values and denser-than-normal bones on roentgenograms, characteristic subperiosteal resorption is still exhibited.

Munson's[20] evidence indicates that the reaction to parathyroid extracts by bone and kidneys can be partially differentiated and is in reality two separate actions. In patients with only a slight tendency to bone lesions, a high calcium diet may prevent their progression, and, if this is true, calcium deprivation should promote bone lesions. Further studies will determine whether bone lesions can be induced in hyperparathyroid patients maintained on a low calcium diet.

SUMMARY

Of a total of 61 patients in whom the surgically removed parathyroid gland proved to be neoplastic on histologic examination, roentgenograms of 47 were adequate for evaluation. Thirteen had roentgenographic evidence of osteitis fibrosa. All of these patients had subperiosteal cortical resorption of some of the middle phalanges of the hands.

Cortical resorption at other sites, such as at the lamina dura and tendinous insertions, was less frequently seen.

Diffuse demineralization of the skeleton was not always visible on roentgenograms even when other roentgenographic evidence of bone involvement existed. The demineralization, when present, was usually of coarse granularity or striated.

Localized destructive lesions ("brown tumors") may be present, even in the jaws, without significant resorption of the lamina dura. All patients with "brown tumors" had subperiosteal cortical resorption of the middle phalanges of the fingers.

The alkaline phosphatase level was significantly elevated in 11 of the 13 patients whose roentgenograms showed evidence of osteitis fibrosa. The extent of osseous disease correlated well with the level of serum alkaline phosphatase, but not with the serum calcium or phosphorus values.

Roentgenographic evidence of urinary calculi was present at one time or another during the course of the disease in 36 patients. Four of these patients had diffuse nephrocalcinosis.

Esophageal or tracheal displacement was observed in 6 of 30 patients who had roentgenograms of the area. This finding was of doubtful value, however, since many patients with hyperparathyroidism also have thyroid neoplasms which may cause displacement.

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ADDENDUM

Since this manuscript was accepted for publication, 34 additional patients with hyperparathyroidism have been studied, making a total of 95. Of the last 34 patients, 1 had two water-clear-cell adenomas of the parathyroid, 1 had endocrine adenomatosis; the other 32 each had single parathyroid adenomas. In the total series of 95 patients, 22 had roentgen evidence of osteitis. In 14 patients, far advanced bone disease with multiple cysts was present; in 7 patients, subperiosteal resorption of bone was the only evidence of hyperparathyroid bone disease; in 1 patient there was no subperiosteal resorption of the pha-

langes, but patchy resorption of the lamina dura was associated with a histologically proved giant cell tumor of the mandible. This was the only 1 of 16 dentulous patients examined in the additional 34 patients who showed any dental abnormalities attributable to hyperparathyroidism. The total incidence of osteitis fibrosa demonstrated on roentgenograms in the entire 95 patients was 23 per cent.

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ROENTGEN CHANGES IN REITER'S SYNDROME

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REITER's syndrome is a triad of urethritis, arthritis, and conjunctivitis. In 1818 and 1836 Brodie[1] reported 6 case histories of men who had this triad of symptoms. In 1916 Reiter[9] described the symptom-complex in patients with dysentery. Accounts of the postdysenteric form of the syndrome, as reported in Europe, indicate that it has the same characteristics and clinical course as the venereal form. The syndrome occurs almost exclusively in the male and is of unknown etiology.

Although the triad of symptoms has been emphasized, many patients also have diarrhea as an early symptom, and others have mucocutaneous lesions. Patients with nonspecific urethritis and arthritis alone have a clinical course indistinguishable from the complete syndrome. Many investigators[2,10] believe that nonspecific urethritis with arthritis is an incomplete form of the fully developed syndrome; therefore, these cases are included in this study. The following features are reported because of their special roentgenologic interest.

Sixty-four patients with Reiter's syndrome, or nonspecific urethritis associated with arthritis, were treated at Walter Reed General Hospital from 1950 to 1960. The available roentgenograms of 46 patients were reviewed to determine what roentgen changes might suggest the syndrome. The evidence collected indicates that the syndrome has a consistent roentgenologic pattern.

ROENTGEN FINDINGS

Acute polyarthritis dominated the clinical picture, although occasionally a single joint was involved. The predilection for the joints of the lower extremities and sacro-iliac joints is found in all large series (Table I).

The frequency and distribution of roentgenographic changes are shown in Table II. Characteristic changes were most frequently found in the bones and joints of the feet, ankles, and in the sacro-iliac joints. The knees were often involved but with no characteristic findings. The hips and shoulders were less commonly involved. In only a few cases were changes in the elbow, wrist, phalanges of the hand, symphysis pubis, and sternoclavicular joint found. Clinical involvement of the feet, ankles, knees, and sacro-iliac joints was associated with roentgenographic changes to a greater degree than in other joints. In many cases the only changes noted were peri-articular swelling and subchondral bone resorption. Other cases progressed within a period of weeks or months to the characteristic changes of periostitis and bony erosion.

Most cases in which severe roentgenographic changes occurred were in patients with an illness lasting several months or those with repeated attacks over a period of many months.

Roentgenograms taken early in the course of the illness seldom showed more than peri-articular swelling and slight subchondral resorption of the adjacent bones. Occasionally, joint effusion was noted, especially in the knee. Some patients respond to treatment and the disease never progresses beyond this phase. When symptoms progressed, with limitation of motion of the involved joints, patchy and frequently marked diffuse demineralization was observed. These findings were most common in the metatarsophalangeal joints, ankles, knees, and interphalangeal joints of the feet and hands. These roentgenographic

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TABLE I

FREQUENCY AND DISTRIBUTION OF JOINTS CLINICALLY INVOLVED

Upper Extremity		Head and Trunk		Lower Extremity	
	Per Cent		Per Cent		Per Cent
Fingers	14	Sternoclavicular	5	Hip	28
Wrist	16	Lumbar and sacro-iliac	42	Knee	67
Elbow	12	Temporomandibular	2	Ankle	65
Shoulder	21			Tarsal and metatarsals	65
				Toes	44

changes were usually reversible. Although some patients showed only the usual reversible changes of peri-articular swelling and demineralization, many had severe clinical joint symptoms with repeated attacks resulting in bony cortical erosion, periosteal new bone formation, and joint destruction. In this chronic stage of the illness, the characteristic fluffy prolific periosteal new bone formation was noted on the plantar surface of the calcaneus. Marginal erosions and loss of the joint space with marked demineralization were noted in some cases, especially in the metatarsophalangeal and interphalangeal joints of the feet. There were no cases of ankylosis, and the only significant deformity observed was the painful calcaneal spur.

Erosion of the articular surfaces was most often seen in the small joints of the feet, knees, and sacro-iliac joint. The earliest erosions were small circumscribed areas of the periphery of the articular surface surrounded by normal appearing bone (Fig. 1*A*). Only rarely did the erosion progress to destroy the subchondral cortex

TABLE II

FREQUENCY AND DISTRIBUTION OF ROENTGENOGRAPHIC CHANGES IN 46 PATIENTS WITH REITER'S SYNDROME

Site	No. of Patients Roentgenographed	Changes
Os calcis	20	Periostitis-10; erosion-3
Ankles	30	Soft tissue swelling-15; periostitis-1; demineralization-13; erosion-4
Tarsals	38	Soft tissue swelling-3; erosion-3; demineralization-13
Metatarsals	38	Soft tissue swelling-10; periostitis-3; demineralization-15; erosion-3
Phalanges of foot	38	Soft tissue swelling-1; periostitis-5; demineralization-14; erosion-4
Hips	10	Demineralization-4; joint destruction-1
Sacro-iliac joints	32	Bilateral changes-11; unilateral changes-4; joint narrowing-9; erosion-15; sclerosis-6
Spine	6	Lateral calcific bridging-1; demineralization-11
Knee	36	Soft tissue swelling-18; periostitis-1; demineralization-11; effusion-8; erosion-4
Shoulder	10	Demineralization-2; erosion-1
Elbow	8	Soft tissue swelling-3; periostitis-2
Carpals	13	Soft tissue swelling-3; erosion-1
Metacarpals	13	No changes
Phalanges of hand	13	Soft tissue swelling-3; periostitis-1; demineralization-2
Symphysis pubis	10	Erosion and sclerosis-2
Sternoclavicular joint	2	Demineralization and erosion-2

FIG. 1. (*A*) Peri-articular swelling about metatarsophalangeal joint with early juxta-articular erosion of base of proximal phalanx of great toe. The joint space is not involved at this time. (*B*) Erosion of metatarsophalangeal joint of left great toe.

of the articular surface except in the sacroiliac and metatarsophalangeal joints (Fig. 1*B*). Only one patient had severe erosion of the hip joint (Fig. 2).

Similar erosive changes occurred on the posterior and inferior aspects of the calcaneus at the attachment of the Achilles tendon and plantar fascia. These changes were always accompanied by pain or tenderness of the Achilles tendon or plantar aspect of the calcaneus. Erosive changes also occurred in the adjacent articular surfaces of the tarsals, malleoli, carpals, shoulders and knees (Fig. 3).

FIG. 2. Unilateral destruction of left hip joint of 22 months' duration.

Periosteal new bone formation was the most characteristic roentgenographic finding. These changes varied with the bones affected. Achilles tendinitis and plantar fasciitis frequently result in loss of associated bony cortex with subsequent bony proliferation into the adjacent soft tissue to produce calcaneal spurs. These spurs were of two types: a simple form in which the margins were well outlined and not associated with changes beyond the base of the spur; and a markedly proliferative form which was irregular in contour. The simple spur is believed to be an ossification in the attachment of the fibers of the long plantar ligament. The characteristic type

Fig. 3. Peri-articular swelling, joint effusion, severe erosion, and periosteal reaction of anterior portion of tibia. Note severe tibiofibular joint destruction.

is a more diffuse process, producing a fluffy outline on the plantar surface of the calcaneal tuberosity and beyond (Fig. 4). Mason *et al.*,[6] in a comparative study of Reiter's syndrome, rheumatoid arthritis, and ankylosing spondylitis, found simple spurs in 40 per cent of the total cases of both Reiter's syndrome and rheumatoid arthritis as compared with 24 per cent in ankylosing spondylitis. On the plantar surface, however, the incidence was significantly different. Periostitis occurred in 20 per cent of the patients with Reiter's syndrome, in 4 per cent of those with rheumatoid arthritis, and in 5 per cent of those with ankylosing spondylitis. They further noted that spurs arising from the posterior aspect were more common in Reiter's syndrome (Fig. 5). In this series the florid type periostitis, often bilateral, was seen in 8 out of 20 patients of whom roentgenograms of the calcaneus were made. The impression that this type of periostitis is probably

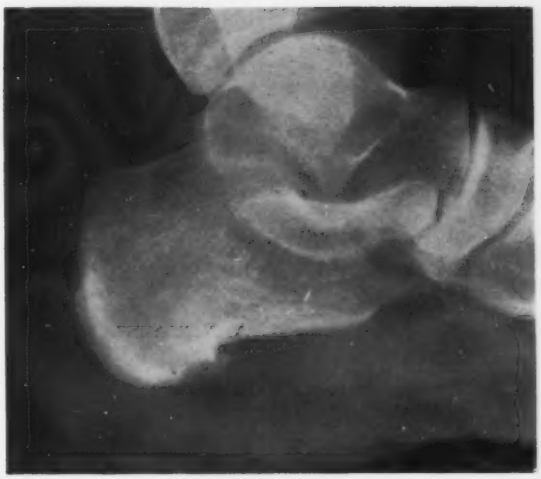

Fig. 4. Characteristic prolific spur on plantar aspect of calcaneus which developed over a period of 9 months.

pathognomonic in Reiter's syndrome is further substantiated.

The second type of characteristic periosteal new bone formation found in this series of Reiter's syndrome was that of linear streaks parallel to the cortex of both the diaphysis and metaphysis of the metatarsals and especially in the toes (Fig. 6). This form of periostitis was also noted in the proximal radius in 2 cases. Occasionally, the periostitis was lace-like in the toes and

Fig. 5. Spur formation on superior aspect of calcaneus at attachment of Achilles tendon.

FIG. 6. Severe demineralization, peri-articular swelling, and linear periosteal reaction about metaphysis and diaphysis of third, fourth and fifth phalanges and metatarsals.

in the shaft of the fingers, much like the type seen in hypertrophic pulmonary osteoarthropathy (Fig. 7, *A* and *B*).

Although early roentgenographic changes in most joints were generally reversible, sacro-iliac joint changes were progressive. A high incidence of sacro-iliitis in Reiter's syndrome has been reported by several authors.[4,5,7,8] Fifteen of 31 patients showed roentgenographic changes in the sacro-iliac joints and these usually occurred in patients with recurrent relapses to which patients with Reiter's syndrome are so prone. The most common finding was narrowing of the joint space with irregular indistinct articular borders and frequent sclerosis (Fig. 8). Seventy-three per cent of the patients showed bilateral roentgenographic changes. Many of the findings were asymmetric.

Neither the clinical nor roentgenologic manifestations of sacro-iliac arthritis are

FIG. 7. (*A*) Marked peri-articular swelling about proximal interphalangeal joints of first, second and fifth digits with periosteal reaction about diaphyses. Note absence of joint involvement. (*B*) Detail of first digit in *A* showing lace-like periosteal reaction of diaphysis of proximal and middle phalanges.

distinguishable with any degree of certainty from those associated with the involvement in rheumatoid arthritis and the invariable bilateral sacro-iliitis in ankylosing spondylitis. There appears to be less extensive destruction and synostosis in Reiter's syndrome than is usually seen in the sacro-iliac joints of ankylosing spondylitis.

Osseous, articular, and ligamentous changes in the spine which are characteristic findings in ankylosing spondylitis were rarely seen in this series of Reiter's syndrome, even when there was destruction of the sacro-iliac joints. This differing incidence of spinal changes is an important roentgenographic feature differentiating these two diseases. Some investigators[3,6] have suggested that classic ankylosing spondylitis may occasionally follow Reiter's syndrome. Only one case in this series would suggest this occurrence (Fig. 9). There is sufficient difference in the mode of onset, clinical pattern, skin and mucosal lesions, and incidence of spinal and peripheral involvement to separate the two as distinct entities.

Roentgenographic changes in the symphysis pubis, indistinguishable from ankylosing spondylitis, are also a recognized feature in Reiter's syndrome. Two such cases were observed in this series.

Murray *et al.*[7] noted that the roentgenographic changes in Reiter's syndrome were similar in many respects to those seen in rheumatoid arthritis and in some cases of ankylosing spondylitis. These authors emphasized that there is a limited number of ways in which a bone or joint can react to a pathogenic agent and that different etiologic factors may cause identical roentgenographic changes.

Fig. 8. Severe bilateral involvement of 18 months' duration of sacro-iliac joints.

Fig. 9. Unilateral calcific bridging on left between L-2 and L-3. Similar findings were noted between T-12 and L-1 on right. Note bilateral early erosive changes in the sacro-iliac joints.

A comparison of the roentgen features of Reiter's syndrome, rheumatoid arthritis, and ankylosing spondylitis by Mason *et al.*[6] stressed that the characteristic florid periostitis in the calcaneus is the only significant differentiating factor, although minor differences in the incidence and distribution occur in other bones and joints. Most investigators have commented on the findings of periosteal new bone formation, es-

pecially in the calcaneus. Others have emphasized osteoporosis. These findings are further confirmed in our series of 46 cases.

SUMMARY AND CONCLUSIONS

The roentgenograms of 46 cases of Reiter's syndrome were reviewed to determine what roentgen features might suggest the diagnosis. The evidence found indicates that this syndrome has a consistent roentgenologic pattern. The high incidence of involvement of the bones and joints of the lower extremity and sacro-iliac is noted. Severe changes were found in patients who had been ill a prolonged period of time or in those who had repeated attacks. The similarities and differences between Reiter's syndrome and rheumatoid arthritis are described. A characteristic fluffy prolific periosteal reaction on the plantar surface of the calcaneus is believed to be specific for Reiter's syndrome.

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TUMORAL CALCINOSIS*

REPORT OF THREE CASES AND REVIEW OF THE LITERATURE

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THE abnormal deposition of calcium salts in the soft tissues is termed calcinosis. Two major types have been generally accepted, namely, calcinosis universalis and calcinosis circumscripta. The former occurs primarily in children and consists of small, hard, calcified nodules and plaques in the skin, subcutaneous tissue and superficial muscle layers. The calcifications are widespread with major involvement of the extremities. Muscle atrophy, contractures, and ankylosis may occur in later stages. The prognosis is generally poor and scleroderma is present in approximately 40 per cent of the cases.

The circumscribed type is usually seen in middle-aged women and has a benign, slowly progressive course. The calcium deposits consist of small nodules and streaks, located primarily about the flexor tendon sheaths in the hands and wrists; occasionally about the elbow and anterior aspect of the knee. Raynaud's disease, scleroderma or sclerodactyly is associated in 30 to 40 per cent of the cases.

A rare pattern of calcinosis, characterized by large, nodular periarticular deposits has been described under various titles.[13,20,22,33,34] In 1943, Inclan[13] introduced the term "tumoral calcinosis." The symptoms, clinical course and roentgenologic aspects do not conform to those described in the universal and circumscribed forms, and recognition of it as a third major type of calcinosis seems justified. Only a few cases have been reported since the publication of Inclan's paper and none have been noted in the American radiologic literature. It is the purpose of this paper to present 3 cases and briefly review the literature.

REVIEW OF THE LITERATURE

Calcinosis has been observed in considerable variety, degree and distribution. Transitional forms and overlap occur, making classification of certain cases into particular types difficult or impossible. However, a majority of the reported cases can be satisfactorily classified as calcinosis universalis or circumscripta. In 1931, Steinitz published a comprehensive review which included 71 cases of calcinosis circumscripta and 34 of calcinosis universalis. In another review, Rothstein and Welt,[27] in 1936, noted 39 cases in children up to fifteen years of age. Of this group, 32 were classified as calcinosis universalis and 7 as calcinosis circumscripta. By 1952, at least 150 cases of the circumscribed form had been reported.[14]

Descriptions of the tumoral type are much less common. In 1935, Teutschlaender[34] used the term lipocalcinogranulomatosis to describe symmetric "calcium tumors" which he observed in a three year old child. He reviewed other cases in the literature which he felt represented the same entity and postulated the pathogenesis on the basis of pathologic findings. He believed that spontaneous fat necrosis was the initial event, followed by calcium deposition and granuloma formation. In his experience lipocalcinogranulomatosis is always found in young people. He regarded it as a rare variety of calcinosis universalis and suspected a hereditary factor.

Sommer and Tress,[33] in 1941, made reference to Teutschlaender and described calcium tumors and chronic renal disease in a forty-three year old female. In addi-

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tion, they mentioned the case of a seventeen year old boy with 5 large calcium deposits, reported by Hoche. Bedetti,[4] in 1946, Oosthuizen *et al.*,[20] in 1950, and Halper,[12] in 1952, reported single cases and referred to Teutschlaender.

Beginning with Inclan in 1943, a series of cases have been reported which were called "tumoral calcinosis." Inclan described the findings in 3 Negroes, aged ten, eighteen and nineteen years. They were not related. Ghormley, in a discussion following the paper, mentions a fourteen year old boy and his 2 sisters with similar calcium deposits. In 1949, Thomson and Tanner[35] added 3 more cases, all Negro male siblings, aged twelve, fifteen and twenty-five years. Palmer,[22] in 1958, reported 2 African males.

Study of the various cases leaves little doubt that Teutschlaender's lipocalcinogranulomatosis and Inclan's tumoral calcinosis are the same entity.

ROENTGENOLOGIC FINDINGS

The calcium deposits are characteristically periarticular and measure from 1 to 20, or more, cm. in diameter. They are dense, irregular and have a round or oval contour. Radiolucent fibrous septa separate clumps of calcium, giving a multinodular appearance. There are no bony abnormalities. The sites of preference are the lateral and superior aspects of the shoulder; the posterior aspect of the elbow and laterally about the hips or buttocks. Other less common sites include the lateral aspect of the feet, and the retroscapular, acromioclavicular, sacral and ischial regions. An occasional deposit has been noted in the neck, and even more rarely, in the hand and in the region of the distal femur. The deposits occupy the subcutaneous and muscle layers. Unless complicated by chronic renal disease or possibly some other abnormality, no other calcification is present. There may be bilateral, symmetric involvement.

CLINICAL FINDINGS

Appearance of the tumors is usually noted in the first or second decade. They are often asymptomatic and slowly increase in size over a period of months or years. Deposits about the hips may not be apparent until they are 8 to 10 cm. in diameter. They are fixed to the underlying tissue, and feel firm and rubbery. There is usually no tenderness, but discomfort was noted in some instances on attempt to move the masses. Occasionally, there is associated pain (Case III), which may be related to a rapid increase in size of the tumor. The overlying skin is stretched but otherwise appears normal except for sites of previous surgical drainage, or for the presence of a fistulous tract when the area has become secondarily infected. Some patients mention trauma to the area prior to the appearance of the tumor, but this history has not been common or consistent.

No limitation of movement is present in the corresponding joint except when very large masses interfere with muscle contraction. The patients have been in good health with few exceptions. One patient reported by Thomson and Tanner[35] eventually expired in a cachectic state and secondary amyloidosis was found on autopsy. Riemenschneider and Ecker[25] reported a single 4×6 cm. deposit in the left lumbosacral region of a fifty-nine year old woman. Associated symptoms of sciatica were relieved by surgical removal of the tumor.

Scleroderma has not been reported in tumoral calcinosis, in contrast to this com-

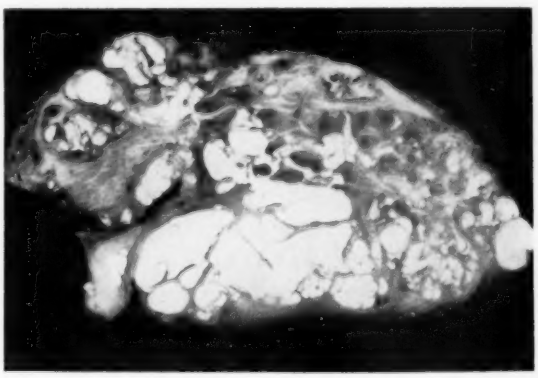

Fig. 1. Case I. Cut surface of the mass removed from the hip. Note the numerous cyst-like cavities filled with pasty, white material. Analysis showed this to be mainly calcium phosphate.

mon finding in calcinosis universalis and circumscripta. The prognosis is good. A number of the patients who have been followed for years did not develop any significant complications.

The presence of tumors in more than one member of a family is a remarkable feature in the small number of reported cases. This is very rare in calcinosis universalis and circumscripta. Laboratory findings are characteristically within normal limits, excluding the patients with chronic renal disease.

PATHOLOGIC OBSERVATIONS

The tumors are firm, rubbery and multilobular with a fibrous tissue covering. When removed, they were found to be firmly fixed by fibrous adhesions to the surrounding muscles, fascia and tendons. The nearby bursae are usually not involved. Sectioning of the tumors reveals a multilocular structure with fibrous connective tissue bands separating pasty, calcareous deposits (Fig. 1). The fibrous walls are lined with multinucleated giant cells, macrophages containing lipoid substance and inflammatory cells (Fig. 2, *A* and *B*). Cultures and stains are negative for any organisms. Analysis of the calcareous material reveals mainly calcium phosphate and has been of no particular help in distinguishing these deposits from those occurring in other forms of calcinosis.

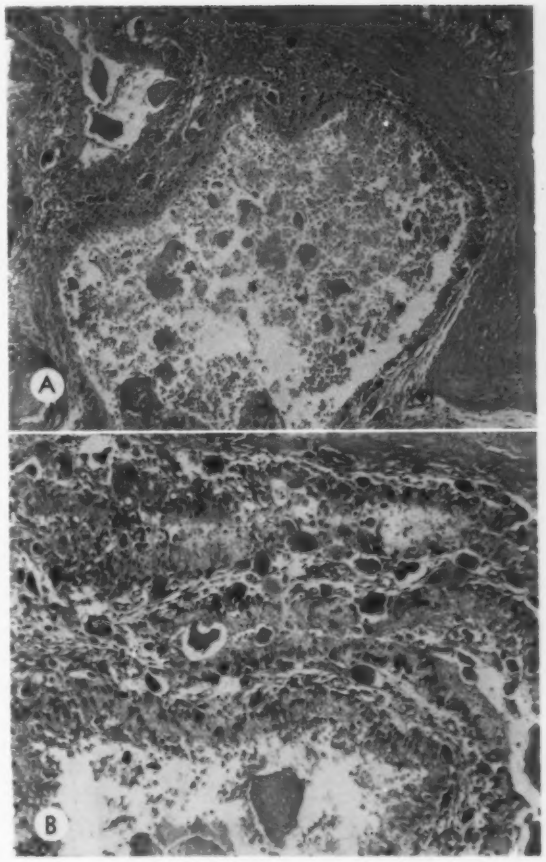

Fig. 2. Case III. (*A*) Section of mass showing large areas of calcification surrounded by dense fibrous tissue (low power magnification 71.5×). (*B*) Many multinucleated giant cells, epithelioid cells, macrophages containing lipoid substances and foamy plasma cells are present (high power magnification 124×).

DIFFERENTIAL DIAGNOSIS

The clinical and roentgenologic findings in calcinosis universalis and circumscripta have already been briefly described. The distinction between these entities and tumoral calcinosis should be possible by both roentgen and clinical features. Occasionally, a case demonstrates more than one pattern of calcinosis. Moore and Clarke[18] reported the case of a forty-six year old female with scleroderma. Roentgenologically, there were features of all three types of calcinosis.

Although metastatic calcification may occur in many diseases, massive periarticular calcification is uncommon. The only entities noted in the literature with calcium deposits similar to those in tumoral calcinosis are in hypervitaminosis D and milk drinker's syndrome. A pertinent history should be obtainable in both of these diseases, as well as the presence of an elevated serum calcium and phosphorus. In addition, other sites of metastatic calcification are usually a prominent feature. The cases of milk drinker's syndrome reported by Poppel and Zeitel[24] showed widespread vascular calcification and deposits in the falx cerebri, kidneys and lungs. In hypervitaminosis D there is usually an associated general decrease in bone density.[8]

Four cases of calcinosis described in patients with chronic renal disease, including

FIG. 3. Case 1. (*A*) The left hip, (*B*) left foot, (*C*) right foot, and (*D*) right elbow. *C* and *D* are common sites of involvement.

Case III, presented here, showed "tumors" which are indistinguishable roentgenologically and pathologically from the cases reported as tumoral calcinosis and lipocalcinogranulomatosis.[6,9,33] One significant difference was in the late onset of the deposits in these patients in contrast to the onset in the first or second decade in the others.

REPORT OF CASES

CASE 1. A twenty-eight year old Negro male was first seen at Duke Hospital in 1945, at the age of thirteen years. A small calcium mass was removed opposite the first metatarsophalangeal joint on the right foot. He was next seen in January, 1960, at which time he had tumoral collections of calcium posteriorly on the right elbow, about the left hip, right lateral aspect of the foot and a 2 cm. nodule over the right acromioclavicular joint (Fig. 3, *A–D*). Physical examination revealed no other abnormalities except for the presence of angioid streaks in both retinae. He stated that the masses had developed slowly over a number of years. His general health was good and he worked regularly as a farmer. There was no limitation of joint movement and no bony abnormalities. He did have recurrent symptoms of secondary infection in the deposit about the hip and this had been drained on two occasions. All of the masses were removed surgically. Pathologically, the findings were the same as those described in previous reports of tumoral calcinosis and lipocalcinogranulomatosis (Fig. 4, *A* and *B*).

Laboratory studies showed a normal urinalysis and hemogram. Three serum calcium and phosphorus determinations were 10.4, 9.8, 10.6

and 6.3, 5.8 and 7 mg. per cent, respectively. Alkaline phosphatase was 1.6 Bodansky units.

Case II. A thirty-two year old Negro male, brother of Case I, was examined when it was learned that he had similar subcutaneous masses removed from the right hip, both elbows and the right foot at the age of twelve years. He had been asymptomatic since that time and was not aware of any recurrence. A roentgen survey did not reveal any deposits at the sites of previous surgery but a 5×9 cm. collection of calcium was present about the left hip (Fig. 5).

Physical examination revealed angioid streaks in the retina, similar to those of his brother, but no other abnormalities. The tumor about the left hip was sufficiently deep to be overlooked. The serum phosphorus was slightly elevated, 5.5 mg. per cent. The serum calcium was 8.5 mg. per cent and other laboratory studies were negative.

Fig. 5. Case II. Roentgenogram demonstrates the asymptomatic calcium deposit. A history of similar deposits about the right hip, both elbows and right foot, removed twenty years previously, was presented.

The father, mother, a sister and another brother were examined and no abnormalities were found.

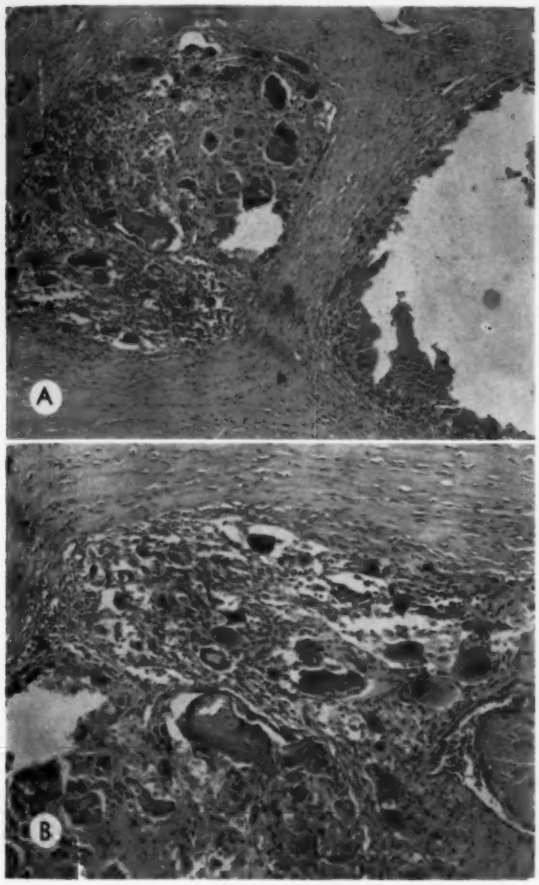

Fig. 4. (*A*) Case I. Section of mass shows dense fibrous bands separating the calcium deposits (low power magnification 71.5×). (*B*) Same cell types as noted in Case III. No bacteria, fungi or other organisms were seen (high power magnification 124×).

Case III. A thirty-two year old thin white female was admitted to Duke Hospital in September, 1957, with a one year history of enlarging subcutaneous masses about both shoulders, elbows, hips and feet (Fig. 6, *A–F*). Roentgenograms revealed these to be dense, multilobular deposits of calcium without any bony abnormalities.

She complained of general malaise, anorexia and a 30 pound weight loss. Periodically, she experienced pain in the region of the masses during the six months prior to admission, when the growth was especially rapid. The overlying skin was stretched, but otherwise appeared normal. Physical examination revealed metastatic calcification in the conjunctiva, cornea, and tympanic membranes bilaterally. In addition, there was bilateral renal calcification and faint vascular calcification in the pelvis.

Excretory urograms showed poor function and phenolsulfonphthalein excretion was 20 per cent in two hours. Serum calcium ranged be-

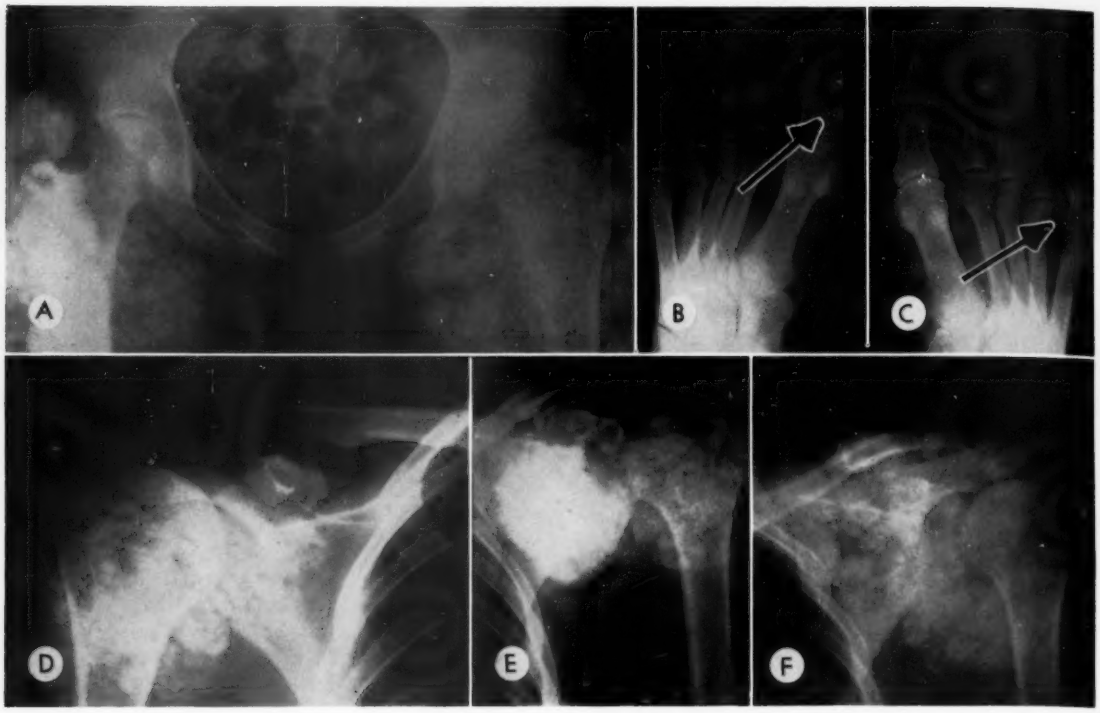

FIG. 6. Case III. Calcium deposits about both hips (*A*), feet (*B* and *C*), and shoulders (*D* and *E*). The massive deposits about the hips extend into the upper thighs. Note in *F*, taken two years after *E*, the increase in deposition about the left shoulder.

tween 10 and 12.6 mg. per cent; serum phosphorus between 4 and 5 mg. per cent. Nonprotein nitrogen was 61 mg. per cent.

Treatment with cortisone and a low calcium and phosphorus diet was instituted but the calcium deposits remained unaltered. On a second admission in March, 1959, a parathyroid exploration was performed. The glands appeared normal at surgery but three were removed. Sections of one gland showed a zone of fibrosis with numerous small tubercles and several multinucleated giant cells. This raised the question of sarcoidosis, but no additional supportive evidence was obtained. A liver biopsy was negative. The serum calcium and phosphorus levels did not change postoperatively.

A portion of the large mass about the left hip was removed in November, 1959. The pathologic findings were the same as those in Case I, (Fig. 2, *A* and *B*).

DISCUSSION

The first 2 cases presented here show the clinical features described in previous reports, namely, an onset in the first or second decade, familial tendency and a benign clinical course. The large mass in the left hip of the younger brother (Case I) became secondarily infected on a few occasions but otherwise the deposits caused no particular discomfort and did not interfere with activity. In a few of the other cases reported, this was also a complication. Case I and II had slightly elevated serum phosphorus levels without apparent cause. This has not been previously reported.

The presence of angioid streaks in the retina in both Case I and II aroused additional interest. Angioid streaks are most commonly associated with pseudoxanthoma elasticum;[28] however, they have also been reported in people with Paget's disease, and recently in 2 cases of sickle cell anemia.[13] The remainder of cases presenting angioid streaks is a small miscellaneous group, including some in which these were reported as incidental findings. Neither Case I nor Case II had observable skin changes and biopsies were negative. It is unlikely that pseudoxanthoma elasticum has any relationship to tumoral

calcinosis. Although vascular calcification is common in pseudoxanthoma elasticum, only one case of the hundreds reported showed soft tissue calcium deposits. This was in a sixty-nine year old white male who had a diffuse deposit of calcium plaques in the soft tissues of the right leg.

The roentgenologic and pathologic findings of the calcium tumors were the same in all 3 cases, but the late occurrence of the tumors and the presence of renal insufficiency with metastatic calcification in Case III are distinctive features. Three other cases have been found in the literature in which tumoral deposits of calcium appeared in people with chronic renal disease.[6,9,33] In these cases the appearance of the tumors was in the fifth decade or later and only after development of renal insufficiency. Although metastatic calcification is common in renal insufficiency of long standing, large periarticular masses of calcium are definitely rare. The relationship between these cases and the younger group who are in good health is not clear.

The pathogenesis of the calcium tumors has not been established. The presence of foamy plasma cells and lipoid substances in the macrophages has attracted most attention, suggesting some origin from fatty tissue. Teutschlaender[34] proposed that the process begins with spontaneous necrosis of fat and this is followed by granuloma formation and calcification. A relationship to the bursae has been considered but in most cases they were not involved. Also, some masses have occurred where there are no bursae.

The only satisfactory therapy has been surgical removal of the masses. The earlier in development that they are removed, the easier and more complete the removal. Although new masses may appear, there is usually no recurrence at the sites of removal and the prognosis appears to be very good.

SUMMARY

Three cases of calcinosis characterized by large, nodular, periarticular deposits are presented. The first 2 cases are Negro siblings who show the typical clinical features, namely, onset in the first or second decade, familial tendency, and a benign clinical course. The third case is an example of calcium tumors in association with chronic renal disease.

This form of calcinosis was described by Teutschlaender[34] in 1935 as lipocalcinogranulomatosis and in 1943 by Inclan[13] as tumoral calcinosis. Because of the use of various terms, the actual incidence is difficult to determine but it appears to be more rare than calcinosis universalis or circumscripta. The symptoms, course and roentgen aspects do not conform to those described in the universal and circumscribed forms and recognition of it as a third major type seems justified.

Dense, nodular masses are seen on roentgenograms, primarily about the hips, shoulders and elbows. Smaller areas may occur adjacent to the spine and about the wrists, feet, lower ribs, and sacrum.

The tumors have a multilocular structure with fibrous connective tissue bands separating pasty, calcareous deposits. Microscopically, macrophages, multinucleated giant cells and chronic inflammatory cells lining the fibrous bands are noted.

Laboratory studies are usually within normal limits and the etiology is obscure. The only effective therapy has been surgical removal of the masses. The prognosis is good. A number of patients who have been followed for years have not developed any significant complications or limitation of activity. The literature is briefly reviewed.

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ARTHROGRAPHY OF THE KNEE*

A CRITICAL STUDY OF ERRORS AND THEIR SOURCES

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MANY papers dealing with arthrography of the knee have already been published. Lindblom[5] in 1948 described his extensive experience in arthrographic procedures and stated the basis for the interpretation of findings as well as his percentage of errors. Various reports have appeared since then. We do not intend to present here another discussion on the principles of arthrographic interpretation; our purpose is merely to analyze this method as a means of diagnosing semilunar cartilage injuries.

The following considerations led to the preparation of this paper: (1) On reviewing the literature, we were impressed by the great differences in arthrographic interpretation by different authors. Whereas the incidence of errors reported by some is as low as 5 per cent (Lindblom[5]), it ranges up to 87.5 per cent in other reports (Oggioni[7]). Obviously, as long as the rate of error does not exceed 5 per cent, this procedure can be considered as an essential aid in the diagnosis of knee lesions. However, errors exceeding this percentage, particularly if they reach as high as 87.5 per cent, will definitely lower its value to a point which does not justify its use. (2) After the great initial enthusiasm for this procedure, there is now a tendency in many clinics to refrain from performing arthrography and, consequently, treatment of knee lesions, including surgical procedures, is performed without its use.

We believe that the situation now requires a thorough re-evaluation of the subject in order to objectively approve or reject the use of arthrography of the knee.

MATERIAL AND METHOD

Since 1957 we have performed 350 arthrographies of the knee using a contrast solution. After a thorough study of the different arthrographic techniques, the positive contrast medium method was accepted in our clinic because the shadow obtained by using air or oxygen was dim in comparison with the sharpness produced by opaque contrast solutions. In addition to this advantage, except for slight swelling in a few cases during the first twenty-four hours, no secondary ill-effects are mentioned in the literature with the use of opaque solutions, whereas air and oxygen are known to cause embolism and synovial reactions. Another complication was reported in 1950 by Murray and Forrai,[6] who observed the appearance of eosinophilia in the synovia and synovial fluid after pneumoarthrography. In spite of these disadvantages, gas is still used as a contrast medium in different clinics (Bonnin and Boldero,[1] and Keats *et al.*[3]). Without dwelling upon further details, and by no means intending to discount the value of a negative contrast medium, we are considering in this report arthrography with opaque chemical solutions only. In all our cases a 35 per cent solution of diodone B.P. was injected. The volume of the solution introduced varied between 8 and 12 cc., depending on the size of the knee.

The investigations are divided into two groups. The first group comprises 150 arthrographies performed using the usual technique; in the second group of 200 studies, some new modifications in technique were introduced. The arthrographies were performed on patients with various complaints in their knees, usually after trauma. In 46 per cent (160 instances) lesions of the semilunar cartilage were more or less evident from the clinical signs. In all the remaining cases the clinical diagnosis was not clear. The age of the patients

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TABLE I

RELATIONSHIP BETWEEN ARTHROGRAPHIC FINDINGS AND ERRORS

	Series I	Series II	Total
Arthrographic Examinations	150	200	350
Arthrographically Diagnosed Lesions	27	93	120
Operations	24	72	96
Errors	5	1	6

ranged from fourteen to sixty years. Twelve were females. Ninety-six patients were operated, and the arthrographic findings were confirmed in 90 instances. The 6 cases with false findings included 5 in our first group: in 3 with negative arthrograms, a tear of the semilunar cartilage was found, and in 2 the positive arthrographic findings were disproved. In the 1 case with false negative findings in our second group, a tear of the medial semilunar cartilage was found (Table I).

We have elaborated on these statistics because they clearly justify the procedures upon which our report is based.

TECHNIQUE

The ordinary technique of injecting a contrast medium into the knee uses the lateral approach above the patellar retinaculum. By using this route the material may penetrate either the upper portion of the joint space or the lower portion of the suprapatellar bursa, which communicate freely with each other and which therefore makes this site more suitable for aspiration than injection. Sometimes, after injection of the material, even when there is no feeling of resistance, the contrast medium does not appear in the joint on the roentgenograms but shows as an irregular patch at the site of the injection. This phenomenon is explained by the fact that the solution may be introduced into the fat body which is located between the bursa and the femur. The result is complete failure of the arthrographic examination in addition to a severe, painful reaction in the knee due to aseptic inflammation of the fat body, which may last for a few days.

In our second series we preferred to use, as an injection site, the medial aspect of the joint, about 3 cm. medial to the patellar ligament when the knee is flexed. At this point the joint space is quite near the skin. The needle penetrates it immediately after puncturing the superficial layers, and the solution is introduced easily and accurately (Fig. 1, *A* and *B*). Flexing the knee subjects the joint to tension, which causes some synovial fluid to leak out through the needle, especially after injecting small amounts of procaine. This is considered to be an indication that the needle is within the joint space. There is only one warning concerning the use of this site: the puncture should be performed at least 3 cm. medial to the patellar liga-

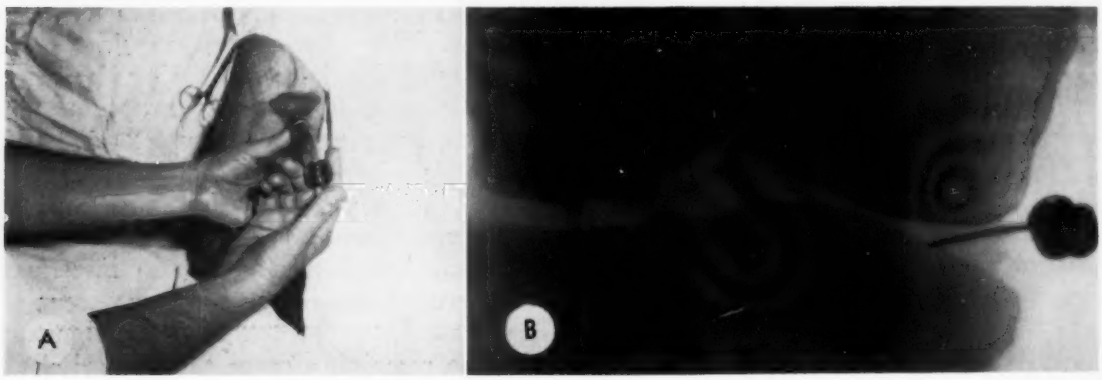

FIG. 1. (*A*) Site of the contrast medium injection at the medial aspect of the joint (when the knee is flexed) about 3 cm. medial to the patellar ligament and (*B*) arthrogram showing position of the needle.

Fig. 2. (*A*) When the puncture is performed near the patellar ligament, the needle penetrates the synovial fold and (*B*) the solution is injected into the fold, outside the joint space.

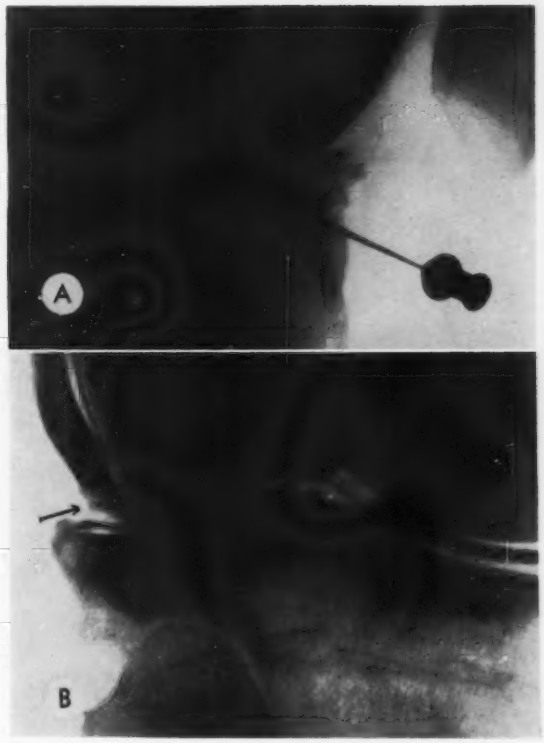

Fig. 3. (*A*) When only a small amount of contrast material is introduced para-articularly, (*B*) in external rotation the artifact (arrow) may give the impression of a lesion of the external semilunar cartilage.

ment, otherwise the needle may penetrate the synovial fold, and the solution will consequently be injected outside the joint space (Fig. 2, *A* and *B*).

If the needle is not held steady during the injection, a very small amount of the contrast material may be injected outside of the joint space (para-articular). In this case the artifact may give the impression of a false lesion of the semilunar cartilage, or it may hide an actual one. Obviously, this is one of the reasons for faulty interpretations (Fig. 3, *A* and *B*).

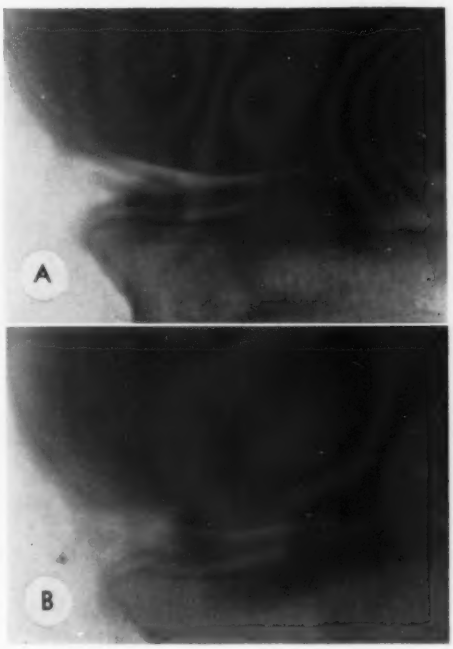

Fig. 4. (*A*) Arthrogram made immediately after injection of the contrast medium shows a tear in the posterior horn of the medial semilunar cartilage. (*B*) Ten minutes later the lesion is not clearly seen.

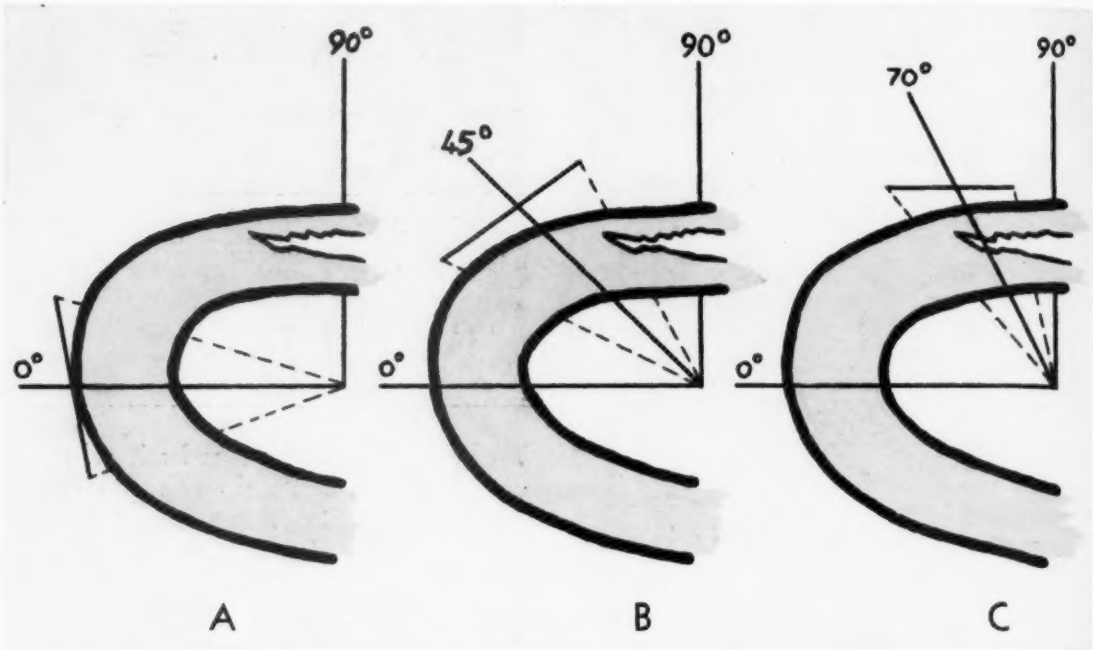

FIG. 5. (*A*, *B* and *C*) Drawings showing the value of increased rotation to reveal lesions in the vicinity of the insertion of the horn. It is obvious that only rotation of 70° will reveal this site (*C*), which will not be shown by the usual rotation of 45° (*B*).

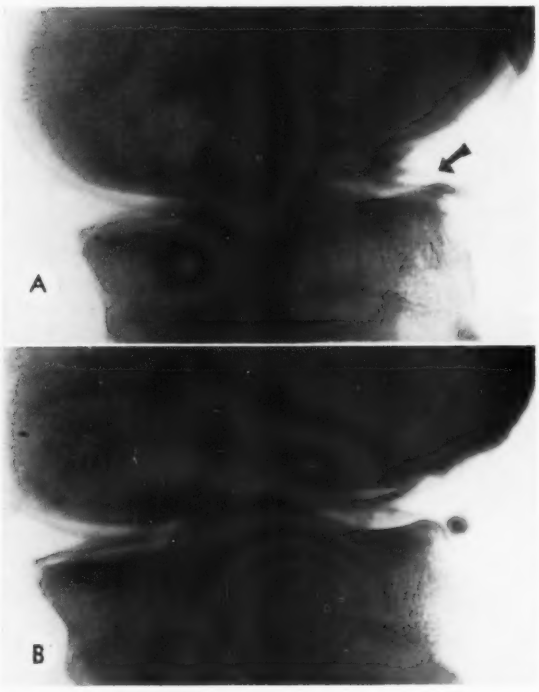

FIG. 6. D.S.95B. (*A*) A lesion (arrow) in the anterior horn of the external semilunar cartilage is demonstrable by 70° rotation. (*B*) It cannot be seen in the usual 45° rotation.

Difficulty in interpretating the arthrograms may be due to the length of time required to perform the whole procedure. In some instances the contrast medium disappears in about half an hour. In others, only dispersed traces of shadow can be seen. For this reason the procedure should take a minimum of time. Any delay will distort the contrast outlines, rendering interpretation difficult (Fig. 4, *A* and *B*.)

Some of the recesses and bursae may cause unusual contrast lines when the knee is examined in different positions. These findings have already been described in many reports and, by studying the interpretation of normal arthrograms, false findings can be avoided.

As is well known, every rotation of the knee reveals only a certain part of the semilunar cartilage. For instance, outward rotation of the knee shows the anterior horn of the lateral semilunar cartilage and the posterior horn of the medial. By increasing the rotation, other areas of the cartilage can be seen. In our opinion, the

usual rotation of 45° is not sufficient to reveal all the possible lesions in the horns. When the lesion is small and is situated in the vicinity of the horn insertion, it can be seen only by increased rotation (Fig. 5, *A*, *B* and *C*). As a standard feature of our procedure, we use a rotation of about 70°; only by this means was it possible to detect an additional number of lesions near the insertion of the horns (Fig. 6, *A* and *B*; and 7, *A*, *B* and *C*).

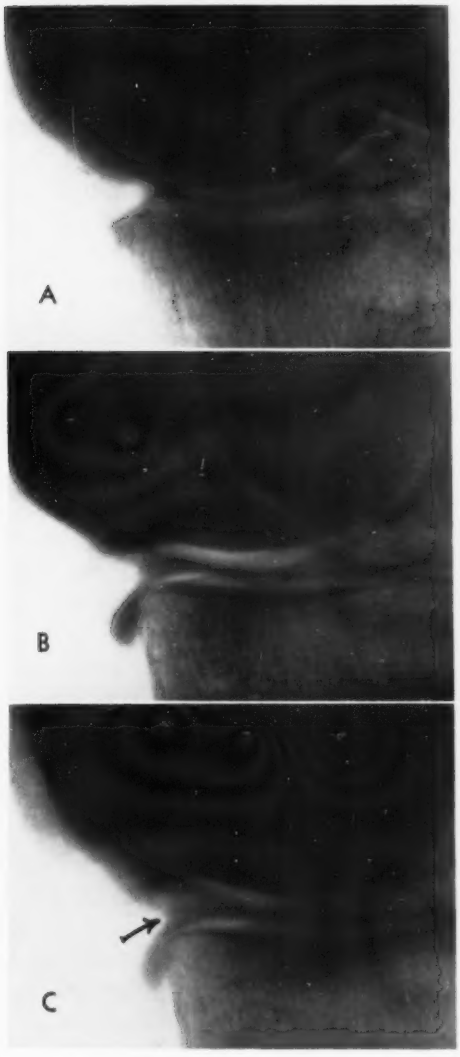

Fig. 7. K.S.73B. Internal rotation of the knee reveals the anterior horn of the medial semilunar cartilage. (*A*) Usual rotation (45°) shows a normal anterior horn. (*B*) With increased rotation (60°) a lesion is suspected. (*C*) With 70° rotation clear penetration of the contrast medium into the tear is revealed (arrow).

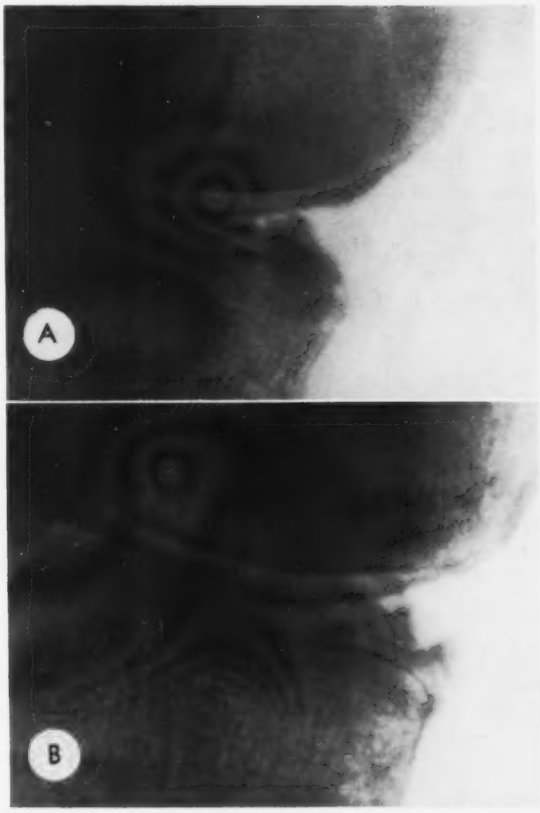

Fig. 8. A.N.65B. (*A*) Cranial projection of 10° reveals a lesion of the posterior horn of the medial semilunar cartilage. (*B*) This cannot be seen in the usual projection.

It is a well-known fact that there are partial lesions which involve the upper or the lower surface of the cartilage without penetrating the whole thickness. In such cases the contrast outlines do not appear smooth but are irregularly thickened at the site of the lesion. Frequently, these thickenings do not appear when the usual projections are used, because parts of the outline are covered by the articular surfaces of the bones. In order to eliminate this factor, a 10°–15° cranial projection has been added, which enlarges the joint space and separates the contrast outlines from the joint surfaces of the bones (Fig. 8, *A* and *B*). It should be remembered that the angle of projection should be carefully selected,

TABLE II

SUMMARY OF FINDINGS BY USUAL AND MODIFIED TECHNIQUES

Lesions	No. of Cases	Per Cent
Diagnosed by Usual Technique	66	70.9
Diagnosed by Maximal (70°) Rotation	17	18.2
Diagnosed by Craniad Projection	10	10.7
Total	93	

otherwise the bones will be superimposed in the opposite direction and the joint space will subsequently be narrowed instead of widened. Lately, laminagraphy has been used to reveal similar lesions (Klami and Kurkipää[4]). This method is rather complicated for routine use, and it is too early yet to evaluate its ultimate results.

RESULTS

As has already been mentioned, the modifications just described were applied in our second series of 200 cases. The results were as follows: of 93 arthrographically visualized lesions, 66 (70.9 per cent) were diagnosed by using the usual, standard technique, 17 (18.2 per cent) were diagnosed only by increasing the rotation, and 10 (10.7 per cent) by adding the craniad projection; in other words, 27 of 93 lesions were not diagnosed by using the accepted technique. This explains 29 per cent of possible errors (Table II). If we add to this the possibility of errors by extra-articular infiltration of the solution, quick absorption, and further unexplained causes, the percentage may reach the figures reported by different authors, as mentioned in the beginning of this paper.

CONCLUSIONS AND SUMMARY

We have not elaborated on the different features of the semilunar cartilages in respect to their anatomic variations as they appear in arthrograms of the knee. This too can be a further cause of faulty interpretation, which can be eliminated by knowing how to correctly evaluate normal arthrograms. This particular phase of the subject has been discussed by different authors.[2,5,8] The purpose of the present report is to analyze knee joint arthrography by describing the different sources of error caused by faulty performance of the procedure and by the lack of sufficient, different projections and positions.

The following points should be observed in order to obtain the best results for evaluation of arthrograms of the knee:

1. Accurate intra-articular injection of the contrast medium is the main factor for successful examination and correct interpretation. A drop of the solution injected extra-articularly, sometimes even when the needle is being extracted, can be a cause of error.

2. Lesions near the insertion of the semilunar cartilage cannot be revealed in the conventional positions. In order to visualize the site of the insertion of the horns, a further rotation of the knee to about 70° should be done.

3. Widening the visualized area of the joint space by using a further cranial projection of 10°–15° is of value in revealing incomplete and superficial lesions of the cartilage. We do not use traction of the leg when performing arthrography, because this does not always result in widening of the space and merely complicates the procedure.

4. Although we mentioned the relationship between the time used in performing the examination and accurate results, we did not elaborate on this particular point, which is a subject for investigation by itself. Our impression is that there are individual variations in the absorption of the contrast medium.

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* Deceased.

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Executive Committee: Milford D. Schulz, Chairman, Boston, Mass.; Theodore R. Miller; Jesshill Love; *Robert L. Brown; *Gilbert H. Fletcher; *Ralph F. Phillips; *Ethlyn Trapp; *Joseph H. Farrow; *Charles G. Stetson.

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Representatives on the American Board of Radiology: Donald S. Childs, Jr., Rochester, Minn.; B. P. Widmann, Philadelphia, Pa.; Frederick W. O'Brien, Boston, Mass.

Representatives on the National Committee on Radiation Protection: Robert Robbins, Philadelphia, Pa.; Elizabeth Focht, New York, N. Y.

Representative on the Board of Chancellors of the American College of Radiology: Milford D. Schulz, Boston, Mass.

Forty-fourth Annual Meeting: Waldorf-Astoria Hotel, New York, N. Y., April 1–4, 1962.

* *Ex officio.*

EDITORIALS

INSTRUCTION COURSES OF THE SIXTY-SECOND ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

PRESIDENT-ELECT Traian Leucutia has requested that we continue presenting the Instruction Courses in connection with the Annual Meeting of the American Roentgen Ray Society to be held in Miami Beach in 1961. These courses originated in 1939 during Dr. Edward Jenkinson's term as President and each succeeding president has recognized the great teaching value of this type of program and has requested its continuance.

It was felt by Dr. B. R. Kirklin, the first director, and by Dr. Harry Weber, his successor, that these courses should be conducted insofar as possible in a relatively informal manner, preferably in small groups. In order to accomplish this, it is necessary to have a large number of courses available. This plan seems like a good one and consequently the program this year will follow the same general plan of the past twenty-one years.

The schedule of the entire program, including brief abstracts of each course, the faculty list, and registration blanks appear in this issue of the JOURNAL. For your convenience the schedule of courses is printed in your program booklet also. It is possible and highly desirable to register in advance for these courses. You are advised to study the schedule of courses, select your first, second and third choice for each day and promptly mail your request to the Director. Since most of the rooms are rather small, many of the courses may be completely filled by the time of the meeting. It will be possible, however, to register at the meeting for any course not already filled. The registration area for these courses will be located in the same section of the hotel as the general registration for the entire meeting. This booth will be staffed Monday afternoon, September 25, and from 8:30 A.M. to 4:30 P.M. Tuesday through Friday, September 29.

The usual division of courses between diagnosis and therapy will obtain. Approximately two-thirds of the periods will deal with diagnostic subjects and one-third will be devoted to radiation therapy, isotopes and physics. There will be many entirely new courses added, including several with particular emphasis on special procedures, such as the one having to do with the management of cardiovascular emergencies. This is designed to alert the radiologists concerning the newest problems, which might arise and must be met in the Department of Radiology. Another extremely important course, given by the Chairmen of three leading Committees and the Chairman of the Board of Chancellors of the American College of Radiology, and moderated by Dr. Earl Barth, its immediate Past President, will deal with medical ethics and economics, radiologic organizations, and various other facts and figures which should be of great interest especially to the younger radiologists. A total of sixty-eight courses will be available, and there is a wide variation of subject matter which should prove stimulating and profitable for everyone.

Most of the courses will be held in rooms on the fourth floor of the Deauville Hotel and three rooms will be available on the mezzanine. All of the rooms are comfortably air conditioned. The courses will start at 3:00 P.M. on Tuesday, Wednesday and

Thursday and at 1:30 P.M. on Friday. Course 305, given by the officers of the American College of Radiology, will start at 1:30 P.M. on Thursday and will be held in Napoleon Room No. 3. *No tickets* will be required for this course.

We feel assured that the educational value of these courses will continue on a very high level and are very grateful to the many instructors who give so willingly of their time and talent.

HAROLD O. PETERSON, M.D.
Director, Section on Instruction

THE FORTY-THIRD ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE American Radium Society held its Forty-third Annual Meeting at the foothills of the Rocky Mountains in Colorado Springs on May 11, 12 and 13, 1961 at the Broadmoor Hotel. The attendance of members and guests eclipsed the previous meeting with a total registration of 502, representing 152 members, 216 non-member guests and 134 ladies. On Wednesday evening, preceding the official commencement of the meeting, the early arrivals were invited to participate in a Western Chuckwagon supper at the famous Flying W Ranch about twenty miles north of Colorado Springs. Three bus-loads of members attended this unique outing in the nearby mountainous ranch country for a delicious barbecued supper and western style musical entertainment with songs from "Wagon Wheels" to "Mountain Dew" by the Wranglers.

The meeting started officially on May 11 in the new parabolically constructed spacious International Center Auditorium. Dr. Juan A. del Regato, Chairman of the Arrangements Committee, welcomed the Society to its first meeting in Colorado Springs. Dr. Jesshill Love delivered the Presidential Address using for his theme the establishment of a centralized radiation center for the treatment of cancer in the smaller communities. He described the organization and facilities of the Memorial Cancer Foundation in Santa Barbara, California, which serves a radius of approximately twenty miles. Emphasis was placed on the importance of engaging a full time radiotherapist for management of the patients and directing the administration while at the same time retaining the principles of free enterprise under a contract with the Board of Trustees of the Foundation and approved by a medical board. All modern facilities are present in this center including supervoltage therapy and an isotope laboratory equipped to perform all useful procedures. Dr. Love hoped that this endowed Foundation in his community would be helpful in serving as a model which might prove instructive and encouraging to other smaller communities.

The first scientific session under the Chairmanship of Dr. Robert L. Brown consisted of presentations on the concept of a cure by Dr. Fred C. D. Collier of Birmingham and a comparative study of supervoltage radiotherapy techniques in some pelvic malignant tumors by Dr. Jose Noriega of the National Cancer Institute of Mexico. A symposium moderated by Dr. Philip Rubin on a critical analysis of the current therapy of carcinoma of the ovary was presented by 6 essayists in an attempt to answer the following questions: (1) What is the natural history of ovarian neoplasms? (2) Is surgery alone adequate? (3) Has postoperative irradiation proven itself? (4) Is preservation of the uterus worthwhile? (5) Of what value are prophylactic isotopes? (6) What is the promise of chemotherapy? The latter part of the morning session was devoted to papers on observations of radioresistant neoplasms and the influence of the time factor on the dose response curve.

A luncheon meeting of members, guests and ladies was held in the International Center Dining Room during which Dr. E. Dale Trout of the General Electric Company delivered an interesting historical talk on the life and work of T. Christen who was the first scientist to use the half-value layer as a means of specifying radiation beam quality.

The afternoon session began with the presentation of experimental work prepared by Dr. Lacassagne of the Institut du Radium of Paris. Because of Dr. Lacassagne's absence, the communication was read by Dr. Juan A. del Regato, the subject being on the effects of the combined actions of x-rays and of a chemical carcinogen on the rat's liver. Dr. Gilbert H. Fletcher, Chair-

man for the afternoon session, called on Dr. Joseph L. Morton to act as moderator for a panel discussion on radiosensitivity and radiocurability as judged by microscopic techniques. Dr. Arthur T. Hertig spoke on the correlation of radiosensitivity and histopathology of cervical cancer. Dr. Ruth M. Graham presented the cytologic prognosis of cancer of the cervix and Dr. Saul B. Gusberg related his experience on radiosensitivity testing of cervix cancer in terms of tumor response. The afternoon session continued with papers on the incidence and treatment of radiation necrosis of the oral cavity and on distant metastases from oral and pharyngeal cancer.

The usual highlight of the Annual Meeting is the Janeway Lecture. Dr. Edith H. Quimby of New York reviewed the life of Dr. Henry Harrington Janeway and introduced Dr. Norman McCormick of Windsor, Ontario, who in turn introduced his fellow Canadian, The Janeway Lecturer, Dr. Clifford L. Ash of Toronto, whose subject was "Oral Cancer—A Twenty-five Year Study." He paid tribute to his former teacher, Dr. Gordon E. Richards and to his surgical colleague Dr. Harold Wookey. Stress was placed on the cooperation between the various specialties in the successful management of oral cancer. He described in some detail the facilities afforded by The Ontario Cancer Institute in association with the Princess Margaret Hospital and Divisions of Biological and Physics Research. During the years 1929 through 1958, 1,837 cases or oral cancer were registered. Statistics for five year results were obtained on 1,624 patients from 1929 through 1955 representing a 96 per cent follow-up. The crude five year survival rates by site and stage were given in detail for cancer of the tongue, buccal mucosa, gingiva, floor of the mouth and palate. The over-all five year results for all sites and stages was 31.5 per cent. The treatment policy is primarily radiotherapy, cervical metastases being managed by radical neck dissection.

Later in the evening of the first day, a reception was sponsored by the Radium Chemical Company of New York on the attractive Terrace Pool of the Broadmoor Hotel. An enjoyable social hour was spent by the members and guests of the Society.

The second day's scientific session under the Chairmanship of Dr. Charles G.Stetson was filled with a variety of presentations. Drs. Chau, Fletcher, Rutledge and Dodd of Houston discussed complications in the high dose total pelvic irradiation of female cancer and Drs. Stander and Rhamey of Indianapolis presented findings on the intravenous pyelograms in patients with carcinoma of the cervix. Dr. Theodore R. Miller of New York City reported on 36 patients who were treated by hemipelvectomy for far advanced cancer. One of the interesting topics of present day research was given by Dr. Robert Rugh of Columbia University on low-level x-irradiation in the very early mammalian embryo. He used exposures of 5 to 15 r on 0.5 day to 1.5 day rat embryos and showed striking central nervous system anomalies. Dr. Edith H. Quimby in collaboration with Dr. John M. Hanford of Columbia University reported on cancer arising many years after radiation therapy of benign lesions in the cervical region. Out of 247 traced patients from a group of 650, there had developed 8 carcinomas of the thyroid, 3 of the pharynx and larynx and 1 of the parotid gland.

The latter part of the morning session included reports on the ten year evaluation of the treatment of bone metastasis from carcinoma of the breast and prostate using radioactive phosphorus and testosterone by Drs. J. G. S. Maxfield and J. R. Maxfield, Jr., characteristics of a cesium head-and-neck unit by Dr. M. Lederman of the Royal Marsden Hospital, the measurement of "build-up" on a curved surface exposed to beams of cobalt 60 and cesium 137 by Drs. R. S. Bush and H. E. Johns, clinical and biological studies of actinomycin D and radiations by Dr. G. J. D'Angio and medical support of air defense operations under radioactive fallout by Captain W. B. Dye, USAF, MC.

During the afternoon of the second day,

a visit was made in buses to the Air Force Academy. A reception was sponsored by the Society at the International Center preceding the Annual Banquet. Many of the past Presidents of the Society were seated at a head table and were introduced by Dr. Jesshill Love. With appropriate ceremony Dr. Clifford Ash was presented the Janeway Medal for his outstanding achievements. Following dinner, a colorful and exciting dance pageant was staged by the Koshare Indians, a group of Boy Scouts who have been well trained in the folk-lore of many ancient tribal dances. This outstanding entertainment was followed by music and dancing for members and guests of the Society.

Dr. Victor A. Marcial was Chairman of the third day's scientific session. A presentation on the Plummer-Vinson syndrome and postcricoid carcinoma was given by Dr. Folke Jacobsson of the Radiumhemmet. A board of inquiry was moderated by Dr. Milton Friedman in which Dr. Fernando G. Bloedorn of Baltimore presented a three year survival of a patient with an extensive peritonsillar carcinoma with metastases to the homolateral lymph nodes, Dr. T. A. Watson of Saskatoon showed a ten year cure of a giant-cell tumor of the cervical spine and Dr. James A. Martin of Dallas demonstrated treatment techniques on an eight year survival of a recurrent low-grade fibrosarcoma of the vulva. Discussion of these cases was given by Drs. del Regato, Ennuyer, Lenz, and Lederman.

A group of papers relating to diagnostic and therapeutic considerations of brain tumors was next on the program. These communications included problems of instrumentation and technique of localization of brain tumors using radioiodinated human serum albumin, radiation therapy of brain tumors, irradiation of meningioma and glioblastoma multiforme. A three-dimensional radium reconstruction device was reported by Dr. Jerome M. Vaeth and a color motion picture on operating room techniques of radium implants was shown by Dr. Fernando G. Bloedorn. The General Electric Company sponsored the appearance of Dr. Bernard Pierquin from the Institut Gustave Roussy in Paris who presented material on laminagraphy in the dosimetric calculations of interstitial curie therapy.

One of the striking statistical reports of the meeting was given by Dr. Sigvard Kaae from the Radium Center in Copenhagen. Five year results on two random series of breast cancer treated by simple mastectomy and postoperative irradiation (McWhirter) versus extended radical mastectomy (Dahl-Iverson) indicated no significant difference. The last paper on the program by Drs. A. Ennuyer and J. P. Bataini on five and ten year results on combined surgery and radiotherapy in the treatment of cancer of the laryngeal vestibule was read by Dr. Juan A. del Regato.

The Forty-third Annual Meeting of the American Radium Society in the attractive setting of Colorado Springs was successful from the standpoints of quality of its scientific program, attendance, good weather and social events. Dr. Jesshill Love and his energetic committees can well be proud of their efforts.

Harry Hauser, M.D.

SAMUEL W. DONALDSON
1891-1961

DOCTOR SAMUEL W. DONALDSON, aged sixty-nine, Chief Radiologist at St. Joseph Mercy Hospital, Ann Arbor, Michigan, died of a heart attack in the early morning hours, Saturday, April 8, 1961.

Doctor Donaldson was born in Rockford, Tennessee, September 14, 1891. His early education was received in private and public schools in Asheville, North Carolina, and Knoxville, Tennessee, and he graduated from Knoxville High School in 1907. Following his graduation from high school, he entered the University of Alabama,

where he remained for one semester. He then entered the University of Tennessee and received his Bachelor of Arts degree in 1912. Entering the University of Michigan, he received his M.D. degree in 1916. Following this he interned at the German Hospital, which is now known as Lenox Hill Hospital, New York City, 1916–1917. He received his radiological training under the late Preston M. Hickey, at the University of Michigan Medical School, Ann Arbor, Michigan.

At the time of his death, Doctor Donaldson was associated with St. Joseph Mercy Hospital since 1923. In addition to his active services as Chief Radiologist in St. Joseph Mercy Hospital, he was also Radiologist at the Ypsilanti State Hospital, Ypsilanti, Michigan, having held that position since 1946. Doctor Donaldson organized the X-ray Department at St. Joseph Mercy Hospital, which he headed for thirty-eight years.

He became nationally known for his contributions in the field of radiology. Throughout his entire medical career as a radiologist, he participated actively in the organizations of radiology with which he was associated. His services and contributions to the advancement of radiology were outstanding. It has been said that "People acquire a varying mental equipment for the journey of life," and this may be said of Sam Donaldson.

He had a particular flair for statistics, and among his hobbies were medical economics and forensic medicine. His statistical knowledge concerning radiological departments, cost per patient, per film, and the personnel required in the radiological departments to handle a given number of patients per day was set forth in a paper which provoked an enormous amount of interest and comment following its publication. Indeed, it is accepted as a manual for the organization of radiological departments. He also published a text on medicolegal practice, which passed through its second edition.

His love for cooking led him to compile eleven cook books in one, called "Ann Arbor Cook Books, 1899–1959."

He maintained a great interest in sports, and his close friends maintained they were sure he could answer the $64,000 question concerning baseball in its various phases and its practice.

Doctor Donaldson pursued many activities, authoring numerous papers in his field, serving as a physician, participating in numerous professional societies, training x-ray technicians, and serving the community with a "side line." It involved x-raying animals as "a courtesy for neighbors who otherwise couldn't get this type of service." He used to say "My veterinary practice has made firm friends for medicine, for radiology, and for doctors."

Doctor Donaldson was the recipient of many honors, among which was a fellowship in the American College of Radiology, and he was awarded in 1955 its Gold Medal for distinguished service in the advancement of radiology.

He was a member of the American Medical Association, American Roentgen Ray Society and at the time of his death was Second Vice-President of this Society, a member of the Radiological Society of North America, the Detroit Roentgen Ray and Radium Society, and the Michigan State Medical Society. At the time of his death he also was Chairman of the Medicolegal Committee of the Radiological Society of North America, Director of the Professional Bureau and a board member of the American College of Radiology. He was a former editor of the Phi Rho Sigma journal. He was a member and Past-President of the Washtenaw County Medical Society, Past-President of the Detroit Roentgen Ray and Radium Society, and Past-President of the Michigan Association of Radiologists.

Doctor Donaldson was a major in the Medical Corps in World War I.

In 1919, he married Kathryn P. Overman of Danville, Illinois. She died in 1954, and he married Margaret L. Lowe of Seattle, Washington, in 1957, who sur-

vives him. He is also survived by a daughter, Mrs. G. B. (Mary E.) Hotchkiss, Washington, D. C., and a son, John G., Madison, Wisconsin, and six grandchildren.

Doctor Donaldson had a host of friends throughout this country and abroad, who will be grieved to learn of his untimely death.

Lawrence Reynolds, M.D.

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

Harold O. Peterson, M.D., *Director*
Donn G. Mosser, M.D., *Associate Director*

Titles and Abstracts of Courses Offered

Sixty-Second Annual Meeting
Deauville Hotel, Miami Beach, Florida
September 26 to September 29, 1961

The Instruction Courses for the Sixty-Second Annual Meeting of the American Roentgen Ray Society will all be of ninety minutes' duration and will be held between the hours of 3:00 P.M. and 4:30 P.M. on Tuesday, Wednesday and Thursday and from 1:30 P.M. to 3:00 P.M. on Friday. No other official activity has been scheduled for this time.

Seventeen rooms are available for the courses and one course will be offered in each room each afternoon of the Annual Meeting. A total of sixty-four instructors will give sixty-eight courses during the four days.

Full information will be available at the desk of the Section on Instruction, which will be located in the General Registration Area on Monday afternoon and from 8:30 A.M. until 4:30 P.M. Tuesday, Wednesday, Thursday and Friday.

How to Register and Obtain Tickets for the Instruction Courses

Admission to the Instruction Courses will be by ticket only, except Course 305.

Following the list of titles and abstracts in this issue of the Journal there is a pink order sheet. First, second and third choices for each period should be indicated on the order sheet. It is possible for one to attend a total of only four periods of instruction. If the directions given on the order sheet are followed explicitly, errors in completing reservations for the courses will be minimized. It is estimated that the number who can attend each course each day will range between 30 and 100 persons depending on room size.

Reservations for the courses will be made in the order in which the pink order sheets are received. Those who are not members of the American Roentgen Ray Society will be charged the nominal fee of $2.00 per period. Full-time graduate students in radiology will be admitted *without fee*, but they will be required to make application for the courses they wish to attend.

Prior to September 12, 1961, the order forms should be sent to the Director, Dr. Harold O. Peterson, 1995 West County Road "B", St. Paul 13, Minnesota. After September 12, 1961 the order forms should be sent to Dr. Harold O. Peterson, Deauville Hotel, Miami Beach, Florida.

To avoid the confusion always existing on the first days of the meeting, a set of tickets will be mailed to each person whose registration for courses reaches the office of the Director on or before September 12, 1961. The envelope containing the tickets will be labelled in a conspicuous manner. Registrants are urged to be on the lookout for their tickets and to bring them to the meeting. The success of this plan depends entirely on the cooperation of the registrants.

In case the courses are not filled by the time of the meeting, tickets will be available at the Registration Desk of the Section on Instruction, Monday afternoon, September 25, and thereafter during the meeting.

Holders of Tickets

Those who do not have the proper ticket for the course they wish to attend will not be permitted to enter the room. An attendant will be at the door of each conference room to collect the tickets.

THE FACULTY

1. Kurt Amplatz, M.D., Assistant Professor of Radiology, University of Minnesota Medical School, Minneapolis, Minnesota.
2. Wilbur Bailey, M.D., Chairman of the Judiciary Committee, American College of Radiology, Los Angeles, California.
3. Robert P. Barden, M.D., Department of Radiology, Chestnut Hill Hospital; Associate Professor, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania.
4. Earl Barth, M.D., Past President of the American College of Radiology, Professor and Head of the Department of Radiology, Northwestern University School of Medicine, Chicago, Illinois.
5. Frederic C. Bartter, Chief of the Endocrinology Branch of the National Heart Institute, Bethesda, Maryland.
6. Frank Batley, M.B., Ch.B., D.M.R.T., Director of Radiation Therapy, Syracuse Memorial Hospital, Syracuse, New York.
7. Fernando G. Bloedorn, M.D., Professor and Head of the Division of Radiotherapy, University of Maryland Hospital, Baltimore, Maryland.
8. Wallace D. Buchanan, M.D., Chairman of the Committee on Hospital Standards, American College of Radiology, South Bend, Indiana.
9. John A. Campbell, M.D., Chairman, Department of Radiology, Indiana University Medical Center, Indianapolis, Indiana.
10. G. J. D'Angio, M.D., X-ray Therapy Department, Children's Hospital Medical Center, Boston, Massachusetts.
11. Joseph N. Debbas, M.D., Department of Radiology, Hahnemann Medical College, Philadelphia, Pennsylvania.
12. Juan A. del Regato, M.D., Director, Penrose Cancer Hospital, Colorado Springs, Colorado.
13. Gerald D. Dodd, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas.
14. Charles T. Dotter, M.D., Professor and Chairman, Department of Radiology, University of Oregon Medical School, Portland, Oregon.
15. Robert L. Egan, M.D., M. D. Anderson Hospital and Tumor Institute, Houston, Texas.
16. Bernard S. Epstein, M.D., Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, New York.
17. Lewis E. Etter, M.D., Director, Department of Radiology, The Falk Clinic; Consultant, Western Psychiatric Institute and Clinic; Professor of Radiology, School of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania.
18. William R. Eyler, M.D., Chairman, Department of Radiology, Henry Ford Hospital, Detroit, Michigan.
19. Benjamin Felson, M.D., Professor and Director, Department of Radiology, University of Cincinnati, Cincinnati, Ohio.
20. Leo S. Figiel, M.D., Department of Radiology, The Grace Hospital, Detroit, Michigan.
21. Nathaniel Finby, M.D., Director of the Department of Radiology, St. Luke's Hospital, Morningside Heights, New York, New York.
22. Harry W. Fischer, M.D., Associate Professor of Radiology, Department of Radiology, University Hospitals, State University of Iowa, Iowa City, Iowa.
23. Robert H. Freiberger, M.D., The Hospital for Special Surgery, Margaret M. Caspary Clinic, New York, New York.
24. Brit B. Gay, Jr., M.D., Department of Radiology, Emory University, Atlanta, Georgia.
25. Bertram R. Girdany, M.D., Director, Department of Radiology, Children's Hospital of Pittsburgh, Pittsburgh, Pennsylvania.
26. John Hale, Ph.D., Associate Professor of Radiological Physics, University of Pennsylvania, Philadelphia, Pennsylvania.
27. Theodore F. Hilbish, M.D., Chief, Diagnostic X-ray Department, The Clinical Center, Department of Health, Education and Welfare, Bethesda, Maryland.
28. John W. Hope, M.D., Director, Department of Radiology, The Children's Hospital of Philadelphia; Professor of Clinical Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.
29. Harold G. Jacobson, M.D., Head of the Division of Diagnostic Radiology of Montefiore Hospital; Professor of Clinical Radiology, New York University College of Medicine; Senior Consultant in Radiology to the Veterans Hospital in the Bronx, New York, New York.
30. John Kirkpatrick, M.D., Department of Radiology, St. Christopher's Hospital, Philadelphia, Pennsylvania.
31. E. C. Klatte, M.D., Associate Professor of Radiology, Chief of Pediatric Radiology, Indiana University Medical Center, Indianapolis, Indiana.
32. Simon Kramer, M.D., Co-Director, Radiation Therapy Department, and Associate Professor of Radiology, Jefferson Medical College, Philadelphia, Pennsylvania.
33. Richard G. Lester, M.D., Professor and Chairman, Department of Radiology, Medical College of Virginia, Richmond, Virginia.
34. Emanuel J. Levin, M.D., Director of Radiology, Maimonides Hospital of Brooklyn; Professor of Radiology, State University of New York, Brooklyn, New York.
35. Alexander Stewart Macmillan, Jr., M.D., Director, Department of Radiology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.
36. Victor A. Marcial, M.D., Director, Cancer Control, Department of Public Health; Director,

Department of Radiotherapy, Hospital Gonzalez-Martinez; Professor of Radiotherapy, University of Puerto Rico Medical School, San Juan, Puerto Rico.

37. Richard H. Marshak, M.D., Associate Radiologist, Mt. Sinai Hospital, New York, New York.
38. Charles L. Martin, M.D., Director, Martin X-ray and Radium Clinic; Clinical Professor of Radiology, Southwestern University Medical School, Dallas, Texas.
39. Gerald M. McDonnel, M.D., Department of Radiology, University of California Medical Center, Los Angeles, California.
40. Harry Z. Mellins, M.D., Professor and Chairman, Department of Radiology, State University of New York, Brooklyn, New York.
41. William T. Meszaros, M.D., Director, Department of Diagnostic Radiology, Cook County Hospital, Chicago, Illinois.
42. Russell H. Morgan, M.D., Radiologist in Chief, The Johns Hopkins Hospital, Baltimore, Maryland.
43. Walter T. Murphy, M.D., Director, Department of Therapeutic Radiology, Roswell Park Memorial Institute, Buffalo, New York.
44. Sidney W. Nelson, M.D., Professor and Chairman, Department of Radiology, The Ohio State University Medical School, Columbus, Ohio.
45. Charles M. Nice, Jr., M.D., Professor and Chairman, Department of Radiology, Tulane University School of Medicine, New Orleans, Louisiana.
46. James J. Nickson, M.D., Chairman, Department of Radiation Therapy, Memorial Hospital, New York, New York.
47. Robert S. Ormond, M.D., Department of Radiology, Henry Ford Hospital, Detroit, Michigan.
48. M. V. Peters, M.D., Ontario Cancer Institute, Toronto, Ontario, Canada.
49. Arthur J. Present, M.D., Chairman of the Committee on Public Relations, American College of Radiology, Tucson, Arizona.
50. Bernard Roswit, M.D., Chief, Radiation Therapy Service, Veterans Administration Hospital, Bronx, New York.
51. Philip Rubin, M.D., Chief, Division of Radiotherapy, Department of Radiology, University of Rochester School of Medicine, Rochester, New York.
52. Eugene L. Saenger, M.D., Associate Professor of Radiology, University of Cincinnati, Cincinnati, Ohio.
53. Jose Sala, M.D., Chief Radiotherapist, Ellis Fischel State Cancer Hospital, Columbia, Missouri.
54. Richard Schatzki, M.D., Chief, Department of Radiology, Mount Auburn Hospital, Cambridge, Massachusetts; Clinical Associate in Radiology, Harvard Medical School, Boston, Massachusetts.
55. William H. Shehadi, M.D., Director, Department of Radiology, United Hospital, Port Chester, New York.
56. Glenn E. Sheline, M.D., Assistant Radiotherapist and Associate Professor of Radiology, University of California Medical Center, San Francisco, California.
57. Edward B. Singleton, M.D., Director, Department of Radiology, St. Luke's and Texas Children's Hospitals, Houston, Texas.
58. Justin J. Stein, M.D., Chief, Radiation Therapy Division and Professor of Radiology, University of California Hospitals, Los Angeles, California.
59. William J. Tuddenham, M.D., Assistant Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.
60. T. J. Wachowski, M.D., Chairman of the Board of Chancellors, American College of Radiology, Wheaton, Illinois.
61. H. Stephen Weens, M.D., Professor and Head, Department of Radiology, Emory University School of Medicine, Atlanta, Georgia.
62. Marvin M. D. Williams, Ph.D., Professor of Biophysics, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.
63. Martin H. Wittenborg, M.D., Children's Hospital; Assistant Clinical Professor, Harvard Medical School, Boston, Massachusetts.
64. George T. Wohl, M.D., Associate Professor of Radiology, Temple University School of Medicine; Chairman, Department of Radiology, Philadelphia General Hospital, Philadelphia, Pennsylvania.

DESCRIPTION OF COURSES

COURSE 101

Tuesday

NATHANIEL FINBY, M.D.
New York, New York

Male Cystourethrography

A general survey of male cystourethrography will stress normal and pathologic roentgenographic anatomy. Congenital and acquired diseases of the lower urinary tract will be demonstrated on cystourethrograms.

COURSE 102
and
COURSE 202

Tuesday and Wednesday

JOHN W. HOPE, M.D.
Philadelphia, Pennsylvania

Roentgen Facts and Fallacies Regarding the Normal and Abnormal Genito-urinary Tract of Infants and Children

Course 102. Probably the most important examination in Pediatric Radiology with the highest yield of positive returns is intravenous urography, and yet, in spite of its importance, it is a study that is frequently poorly executed. There are a multitude of reasons for this. The speaker will attempt to show his own problems with this procedure and how, by working at it over a long period of time, he arrived at a technique which nine out of ten times results in a diagnostic study. This technique will include various methods of reducing the roentgen exposure to the gonads. It will also illustrate the importance of obtaining a voiding urethrogram in the male as one of the final steps in the procedure.

Having presented the technique of intravenous urography, the speaker will then attempt to illustrate his own evolutionary understanding of posterior-urethral valves, ectopic ureteroceles, absence of the abdominal musculature, and of as many other congenital anomalies as time will permit.

Mediastinal Mass Lesions in Infants and Children

Course 202. Mass lesions occurring in the mediastinum can be diagnosed with a high degree of accuracy simply by knowing whether they occur in the front, in the middle or in the back. This course will attempt to give a comprehensive discussion of most of the mass lesions which occur in the mediastinum. There are a few differences between the younger age group and the older age group and these differences will be discussed.

COURSE 103

Tuesday

RUSSELL H. MORGAN, M.D.
Baltimore, Maryland

Screen Intensification and Cineradiography

This course will deal with the physics and technical problems associated with screen intensification and cineradiography. The discussion will consider those aspects of x-ray image formation and perception which are important in the fields of screen intensified fluoroscopy and motion picture recording. Particular attention will be given to the practical problems associated with the use of various intensifier and cineradiographic systems in clinical radiology.

COURSE 104

Tuesday

PHILIP RUBIN, M.D.
Rochester, New York

The Development of a Dynamic Classification of Bone Dysplasias

There is a compelling logical order to the way normal bones model. From a study of the development of normal bone growth and an analysis of the physiologic processes, one may be able to provide the key to unlock the abnormal pathophysiology in dysplastic bone diseases. An attempt is made to set up criteria for true bone dysplasias, based on an understanding of experimental and clinical pathophysiology. It is hoped that one can develop, on this basis, a dynamic classification for this seemingly complicated group of diseases. The differential diagnostic points of different bone dysplasias will be emphasized.

COURSE 105

Tuesday

WILLIAM J. TUDDENHAM, M.D.
Philadelphia, Pennsylvania

New Concepts in Radiographic and Interpretative Techniques

This course consists of an analysis of the sources of error in roentgen diagnosis, a critique of existing concepts of "film quality," and proposals for modified radiographic and interpretative techniques designed to increase the accuracy of the method.

The inadequacy of "contrast" and "unsharpness" as criteria for evaluating the diagnostic usefulness of roentgenograms will be discussed and new criteria will be presented.

The significance of physical factors said to limit the "quality" of roentgenograms will be considered and the indications for so-called "high-kilovoltage" and "supervoltage" radiography as well as for the

use of the "micro-focus" roentgen-ray tube will be specifically noted.

Psychophysiologic factors which limit the accuracy of roentgen diagnosis will be emphasized, and recent experimental studies relating to problems of visual search and image organization will be presented. Also, control of view-box and "surround" illumination, control of glare, and the selection of reading lenses—practical aids for the radiologist in perceiving critical "threshold" images—will be presented.

Pertinent experimental findings will be cited, but the practical clinical application of these concepts will be emphasized through the presentation of illustrative diagnostic roentgenograms.

COURSE 106
and
COURSE 206

Tuesday and Wednesday

LEWIS E. ETTER, M.D.
Pittsburgh, Pennsylvania

Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses

Course 106. Description of standard techniques and positions with illustrative slides for roentgen examination of the skull and paranasal sinuses. Comparison of advantages of one projection over another with respect to visibility of particular structures. General considerations with reference to the cerebral and facial components of the cranium and various skull types. Analysis of individual bone components in producing the various lines and shadows seen in plain roentgenograms of the skull. This analysis will be applied to a study of the sphenoid, frontal, ethmoid, parietal and occipital bones in various projections.

Course 206. Detailed roentgen anatomic analysis of the paranasal sinuses and mastoids with description of roentgenographic techniques employed. Study of the roentgen features of the middle cranial fossa and their relation to the sphenoid sinuses. Consideration of the orbits and optic foramina with special attention to the frontal, ethmoid and sphenoid bone components in various projections as well as the features contributed by the zygomatic bones. In conclusion, the maxillofacial components of the cranium will be examined, including the lacrimal canals and soft tissue delineation.

COURSE 107

Tuesday

H. STEPHEN WEENS, M.D.
Atlanta, Georgia
BRIT B. GAY, JR., M.D.
Atlanta, Georgia

Roentgenology of the Pulmonary Circulation—Diagnosis of Certain Physiologic Changes

I. Methods of roentgenologic study of pulmonary circulation: (a) roentgenoscopy with image amplification, (b) routine roentgenography, (c) laminagraphy, (d) pulmonary angiography and (e) cineroentgenography.

II. Basic function of pulmonary circulation: normal pulmonary arterial and venous pressure; normal pulmonary blood flow.

III. Increased pulmonary blood flow. Definition. Etiology: left to right shunts; admixture lesions. Roentgenologic signs. Pulsations of the pulmonary arteries in increased pulmonary blood flow.

IV. Decreased pulmonary blood flow. Definition. Etiology: left to right shunts with pulmonary stenosis or atresia. Roentgenologic signs. Roentgenologic appearance of collateral circulation to the lungs through bronchial arteries or anomalous arteries. Pulsations of pulmonary arteries in decreased pulmonary blood flow. Special consideration of pulmonary vasculature changes and pulsations in isolated pulmonary valvular stenosis.

V. Pulmonary venous hypertension—pulmonary edema. Roentgenologic signs. Significance of Kerley's (septal) lines. Special consideration of pulmonary venous changes in mitral stenosis. Importance of minimal signs of pulmonary interstitial edema as an indication of acute myocardial infarction.

VI. Pulmonary arterial hypertension. Definition. Etiology: roentgenologic signs. Pulsations of pulmonary arteries in pulmonary hypertension.

The roentgenologic changes of the pulmonary circulation will be illustrated by cinefluorograms.

COURSE 108
and
COURSE 208

Tuesday and Wednesday

EMANUEL J. LEVIN, M.D.
Brooklyn, New York

The Fundamentals of Bone Disease

There are characteristic radiologic patterns produced by diseases of the bones which permit a definite and accurate diagnosis. The reactions in the soft tissues, periosteum and cartilage are often of greater differential value than the changes in bone. Furthermore, many osseous diseases have peculiarities of destruction, production and localization which in themselves are of diagnostic significance. The de-

tection and correct assessment of the varied roentgen patterns will permit an accurate diagnosis in a high percentage of cases.

An analytic approach to the differential diagnosis of diseases of the bones will be presented with emphasis placed on fundamental aspects.

I. Explanation and description of terms
   A. Soft tissue reactions
   B. Periosteal reactions
   C. Osseous reactions
II. Tumors
   A. Benign
   B. Malignant
III. Infections
   A. Acute
   B. Chronic

COURSE 109
and
COURSE 209

Tuesday and Wednesday

ALEXANDER STEWART MACMILLAN, JR. M.D.
Boston, Massachusetts

Sinuses; Mastoids, Lateral Neck and Larynx

Course 109. Sinuses.
Course 209. Mastoids, lateral neck and larynx.

COURSE 110
and
COURSE 210

Tuesday and Wednesday

SIDNEY W. NELSON, M.D.
Columbus, Ohio

Bronchography

Course 110. The radiologist who can properly perform and interpret bronchograms is in a position where he can make outstanding contributions to the diagnosis and understanding of many chest diseases. The proper therapy for any disease which affects the bronchial tree is often dependent upon the accurate determination of the nature and extent of the bronchial involvement. Inasmuch as the interpretation of bronchograms requires considerable knowledge of the normal anatomy of the tracheobronchial tree and some of the common anatomic variations, the pertinent anatomy will be discussed and illustrated. A good knowledge of bronchial anatomy will enable the radiologist to systematically identify each of the major segmental bronchi, because of his knowledge of the expected distribution of each of these bronchi. The identification of each of the major segmental bronchi is extremely important in interpretation.

Once the basic bronchographic anatomy has been considered, the details of the bronchographic technique will be described. This will include the method of performing topical anesthesia, introduction of intranasal catheter, method of injecting contrast medium in order to assure systematic filling of the desired segmental bronchi, proper positioning of patient for filling of each segment, and the spot filming techniques employed to thoroughly record the filling of the desired segmental bronchi. This portion of the discussion will also include a description of some of the characteristic features of various contrast materials. The advantages of fluoroscopically controlled spot filming in comparison with conventional large film techniques will be emphasized. Good bronchographic technique can be mastered with little effort and can be quickly performed by the radiologist and his technical staff, thus precluding the necessity for resorting to the "team" approach which is often so time-consuming and frustrating in large busy hospitals.

Course 210. During the second session, the majority of the different types of bronchopulmonary pathology will be discussed including the following: 1. Chronic bronchitis: a. spasm; b. excessive secretions; c. dilated mucosal glands; d. destruction of mucosa. 2. Emphysema. 3. Segmental bronchiectasis. 4. Cystic lesions. 5. Bronchogenic carcinoma. 6. Pulmonary "coin" lesions. 7. Bronchial adenoma. 8. Erosion of bronchi by adjacent calcified lymph nodes. 9. Inflammatory strictures. 10. Bronchial relocation following atelectasis of surgical removal.

The value of bronchography in the differential diagnosis of many bronchopulmonary diseases will be emphasized throughout the course, as for instance, atelectasis due to bronchogenic carcinoma or other intrabronchial lesions. Segmental bronchiectasis with associated chronic pneumonic changes may mimic neoplasms on plain chest roentgenograms, but can be clearly identified by bronchographic methods. Chronic granulomas and other infections can frequently be identified by virtue of the bronchiectatic changes produced in adjacent bronchi, thus helping to differentiate these diseases from neoplastic processes. Certain pulmonary diseases have very specific bronchographic appearances; *e.g.*, bronchial adenomata, polycystic lung, etc.

It is hoped that this presentation will encourage radiologists to perform this extremely useful roentgenographic procedure more frequently in the future. In so doing, they will be able to make more significant contributions to the diagnosis and treatment of the patients referred to them for study by their colleagues who are not radiologists.

COURSE 111

Tuesday

MARTIN H. WITTENBORG, M.D.
Boston, Massachusetts

Pelvis and Hips in Infancy and Early Childhood

Fundamentals of the roentgen interpretation of the growing pelvis and hips will be presented and illustrated as they apply in the diseases commonly encountered in this age group.

COURSE 112

Tuesday

ROBERT L. EGAN, M.D.
Houston, Texas

Over Five Years' Experience with Mammography

Over five years' experience has been gained with soft tissue roentgenography of the breast in a tumor institute with an active breast service. These studies have proved a highly accurate and valuable adjunct to the clinical and pathologic evaluation of breast lesions.

A simple and readily adaptable roentgenographic technique will be presented. Cases illustrative of typical benign and malignant disease will be shown and the diagnostic features of each described and tabulated. Differential diagnosis, sources of error, limitations of the procedure and the clinical and pathologic implications of the method will be discussed.

The results of a statistical analysis of the first 1,000 consecutive examinations (all with a follow-up from one to five years) will be reviewed. The error in 245 cases of malignant disease was 0.82 per cent.

COURSE 113
and
COURSE 310

Tuesday and Thursday

RICHARD H. MARSHAK, M.D.
New York, New York

The Small Intestine

Course 113. 1. Technique, including a discussion of the various barium preparations. 2. Regional enteritis. 3. The malabsorption syndrome, including sprue, lymphosarcoma and Whipple's disease. 4. Problems in the interpretation of functional changes. 5. Inflammatory lesions of the duodenum, excluding duodenal ulcers.

The Small Intestine and Colon

Course 310. A-1. Infarction of the small bowel. 2. Tumors. 3. Collagen diseases.

B-1. Inflammatory lesions of the colon.

COURSE 121

Tuesday

JUAN A. DEL REGATO, M.D.
Colorado Springs, Colorado

El Papel de la Radioterapia en el Tratamiento del Cancer

(This Course Will Be Given in Spanish)

La cirujía y la radioterapia tienen indicaciones precisas en el tratamiento del cáncer en las que una ú otra son el tratamiento de elección.

Además, existen ciertas posibilidades de fructrosa asociación de ambas disciplinas en beneficio del enfermo.

La radioterapia, como tratamento paliativo de enfermos incurables es, de por sí, un método indispensable en el tratamiento de estos enfermos.

COURSE 122

Tuesday

EUGENE L. SAENGER, M.D.
Cincinnati, Ohio

Diagnostic Uses of Radioisotopes (Other Than for Thyroid Disease)

This course will discuss the clinical uses of radioisotopes utilizing a scintillation probe, a scintillation well counter, a scanner and single channel pulse height analyzer.

The topics to be included are as follows:

I. Brief remarks on standardization of the instrument
II. Hematologic tests
  A. Iodinated albumin
  B. Chromium 51
    1. Red cell mass
    2. Red cell life span
    3. Sequestration
  C. Iron 59
    1. Iron turnover
    2. Iron utilization
  D. Simultaneous techniques utilizing Cr^{51} and Fe^{59}
III. Vitamin B_{12} (Co^{60})—Pernicious anemia
IV. I^{131} labeled fats and fatty acids
V. P^{32} in detection of eye tumors
VI. The scanogram
  A. Liver
  B. Chest
VII. I^{131} labeled PVP for evaluation of intestinal protein loss

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COURSE 123

Tuesday

MARVIN M. D. WILLIAMS, Ph.D.
Rochester, Minnesota

The r, rad and rem in Specification of Dose

Three different units are used in expressing dosage in radiation therapy. These are the exposure dose, which is measured in r, the absorbed dose, which is measured in rads, and the biologic dose, which is measured in rems. The meaning of these three units, their use, and methods of measuring and calculating them will be discussed.

At the present time, sufficient data are not available to calculate dosage in rems. To calculate the dosage in rads involves the use of conversion factors that often are uncertain. The determination of the exposure dose, which is the basis for calculating the absorbed dose, may have a rather high probable error. The combination of these uncertainties and probable errors often makes a statement of dose in rads of questionable value.

COURSE 124

Tuesday

G. J. D'ANGIO, M.D.
Boston, Massachusetts

Radiation Therapy in the Management of Children with Tumors

The discussion will include a description of the most common pediatric-age neoplasms with emphasis on their peculiarities and the unpredictable clinical course followed by some of these tumors. Treatment as practiced at the Children's Hospital Medical Center will be reviewed as will the results of therapy and the late effects of irradiation delivered to infants and children.

COURSE 201

Wednesday

HARRY W. FISCHER, M.D.
Iowa City, Iowa
GERALD D. DODD, M.D.
Houston, Texas
JOSEPH N. DEBBAS, M.D.
Philadelphia, Pennsylvania

Lymphography

The historic development of lymphography will be briefly described. The various techniques which have been utilized for lymphography will be discussed and recommendations made for the best available technique at the present time. Since visualization of the lymphatic structures depends upon specialized characteristics in contrast material, the presently available materials will be commented upon and their advantages and deficiencies compared. Suggestions toward the development of a better contrast material will be made. The anatomy of the normal lymphatic structures will be then described as a base for the discussions of pathologic findings which will follow.

The use of lymphography in diagnosing and evaluating the extent of neoplastic disease in the lymph nodes will be discussed by Dr. Dodd based on his clinical experiences with the method at the M. D. Anderson Hospital and Tumor Institute. The use of lymphography and evaluation of the edematous leg will be discussed by Dr. Debbas.

A summary of the clinical experience will then be made with additional comments on the possible therapeutic applications of the same technique used in the diagnostic field. Time will be allowed for discussion between the three major participants and for questions from the floor.

COURSE 203

Wednesday

CHARLES M. NICE, JR., M.D.
New Orleans, Louisiana

Roentgenographic Manifestations of Collagen Diseases

Many conditions have been included under the term of collagen diseases. In general, diseases involving wide-spread changes in connective tissue or in small vessels have been called collagen diseases at one time or another.

Most writers include systemic lupus erythematosus, polyarteritis nodosa, systemic scleroderma, dermatomyositis, and rheumatic fever in the collagen disease group. Rheumatoid arthritis is considered by many to be a phase of the development of the major collagen diseases described above.

The roentgenographic findings in the above conditions will be described, the material being basically related to a study of 109 patients at the University of Minnesota Hospitals, later followed by an extension of this study at Charity Hospital in New Orleans. Combinations and sequences of findings that will help one to suspect collagen diseases will be emphasized in the presentation.

COURSE 204
and
COURSE 304

Wednesday and Thursday

HAROLD G. JACOBSON, M.D.
New York, New York

Malignant Bone Tumors—Primary and Secondary

Course 204. In this presentation the roentgen characteristics of the important malignant bone tumors—both primary and secondary—will be illustrated. These will include: (1) osteogenic sarcoma, (2) Ewing's sarcoma, (3) chondrosarcoma, (4) sarcoma with Paget's disease, (5) fibrosarcoma, (6) chordoma, (7) synovioma, (8) myelomatosis, (9) reticulum cell sarcoma, (10) Hodgkin's disease, (11) lymphosarcoma, (12) leukemia, (13) metastatic carcinoma.

In all instances the roentgen findings will be evaluated against the background of a discussion of the underlying pathologic changes for an adequate understanding of these roentgen abnormalities.

The Vaterian Segment: Normal and Abnormal

Course 304. The roentgen anatomy of the normal vaterian structures *in vitro* will be described utilizing cadaver material. The *in vivo* appearance of the normal vaterian papillae in the upper gastrointestinal tract examination will be illustrated and emphasized after a brief description of the various methods of the roentgen examination of the vaterian region. There will then follow the discussion of some of the important disease entities involving the vaterian area. The section on abnormalities will be dealt with against the background of a classification of enlargement of the papilla of Vater divided into two major categories: centric and eccentric enlargements. The centric enlargements will include those associated with common duct stone, acute and chronic relapsing pancreatitis, and carcinoma of the papilla and ampulla of Vater. The eccentric enlargements will be demonstrated mainly by cases with peptic ulcer disease in the proximal duodenum. The differential diagnosis of the various disease states causing these enlargements will be emphasized and illustrated with the aid of an outline long in use for evaluating abnormalities of the vaterian segment. The roentgen findings in carcinoma of the head of the pancreas will also be briefly described, particularly as these changes relate to the vaterian segment.

COURSE 205
and
COURSE 405

Wednesday and Friday

CHARLES T. DOTTER, M.D.
Portland, Oregon

The Mechanism and Practical Management of Cardiovascular Emergencies Likely To Arise in a Radiology Department

In association with contrast cardiovascular visualization studies, myocardial anoxia may lead to cardiac arrest or ventricular fibrillation. Unless serious coronary occlusion, aortic stenosis or insufficiency is present, there is an excellent chance of effective resuscitation.

Cardiac arrest. This is most commonly encountered in association with general anesthesia and carries a better prognosis than does ventricular fibrillation. The treatment is prompt adoption of measure promoting an increased flow to the myocardium of oxygenated blood. Mouth-to-mouth resuscitation and anteroposterior compression of the lower sternum against the spine provide effective means for accomplishing this purpose. If a normal heartbeat does not return, the use of a pacemaker may be considered. Thoracotomy is not only unnecessary, it is far less likely to result in survival of the patient.

Ventricular fibrillation. Ventricular fibrillation is prone to occur in people with diminished coronary flow. When it occurs as a product of uncorrected myocardial anoxia, defibrillation will not be successful. Often ventricular fibrillation following coronary occlusion is due to excitation of a small infarct rather than diffuse myocardial anoxia. Here, prompt resuscitation is very likely to be lifesaving. Resuscitation consists of properly applied compression of the lower sternum against the vertebral column. This form of resuscitation is simple to perform and—as will be demonstrated by pressure records comparing both closed and open techniques in the same patient—more effective than direct open-chest cardiac massage. Details will be cited covering several emergency resuscitations including four open-chest cardiac massages with two survivals and one protracted resuscitation wherein left ventricular function was provided by several members of the Radiology Department during a two and one-half hour period. A simple, low-cost (homemade) defibrillator was repeatedly employed with success.

Credit for the technique for closed-chest cardiac resuscitation properly goes to Drs. Kouwenhoven, Jude and Knickerbocker of Johns Hopkins University. This technique will become standard and should, therefore, be familiar to all physicians.

COURSE 207
and
COURSE 307

Wednesday and Thursday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

Some Fundamentals in Chest Roentgen Interpretation

Chest roentgen interpretation should not merely represent an attempt to correlate particular shadows with specific diseases, but should be approached with an understanding of the basic principles of anatomy, physiology, pathology, and radiology involved. Some of these principles will be considered and their practical application illustrated.

Among the subjects to be discussed are:

Course 207: (1) A method of fluoroscopy; (2) the "silhouette" sign; and (3) the air bronchogram.

Course 307. (1) Anatomic variations in the pulmonary fissures; (2) lobar collapse; (3) lobar enlargement; and (4) the disrupted fissure.

COURSE 211

Wednesday

RICHARD SCHATZKI, M.D.
Boston, Massachusetts

Differential Diagnosis of Lesions at the Lower End of the Esophagus

The technique of the examination of the lower end of the esophagus will be described. Some of the problems in this area will be discussed, particularly those concerning esophagitis, cancer, lower esophageal ring and hiatus hernia.

COURSE 212

Wednesday

WILLIAM R. EYLER, M.D.
Detroit, Michigan
ROBERT S. ORMOND, M.D.
Detroit, Michigan

The Acute Abdomen

This course is designed primarily to review the roentgenologic manifestations of acute abdominal disease, particularly, as manifested on plain abdominal films. When appropriate for illustrative purposes, associated examinations with contrast materials will be presented. The following outline includes the subjects to be discussed:

1. Anatomy of the peritoneal cavity as shown by pneumoperitoneum.
2. Differentiation of the hollow viscera.
3. Vascular diseases: (a) aneurysms, dissecting and arteriosclerotic; (b) occlusive vascular disease; (c) blood dyscrasias; (d) polyserositis; (e) collagen diseases.
4. Inflammatory diseases of hollow viscera: (a) acute appendicitis, acute cholecystitis, gastritis, enteritis and cholecystitis; (b) regional enteritis; (c) tuberculosis; (d) generalized peritonitis; (e) localized abscesses.
5. Inflammatory diseases of solid viscera: (a) acute pancreatitis; (b) acute perinephric and obstructive urinary tract diseases.
6. Mechanical obstructions of the digestive tube: (a) neoplastic diseases; (b) extrinsic pressure; (c) special mechanical obstructions including intussusception, volvulus, hernia and band.
7. Traumatic conditions: (a) solid viscera; (b) hollow viscera.
8. Miscellaneous conditions causing or simulating the acute abdomen including sprue, neurologic disorders, toxic agents and endocrine malfunctions.

COURSE 221

Wednesday

GLENN E. SHELINE, M.D.
San Francisco, California

The Use of Radioisotopes in the Diagnosis and Treatment of Thyroid Disease

This course will deal with the uses of radioiodine in the diagnosis of thyroid dysfunction, in the treatment of thyrotoxicosis, and in the treatment of thyroid cancer. In the material on diagnosis, emphasis will be placed on the thyroid uptake curve with or without thyroid suppression or stimulation tests. The uses and limitations of thyroid scanning will be considered. Included in the treatment of thyrotoxicosis will be information regarding the occurrence of leukemia, thyroid adenomas and thyroid carcinomas subsequent to radioiodine therapy.

COURSE 222

Wednesday

JUSTIN J. STEIN, M.D.
Los Angeles, California

The Use of Megavoltage Therapy in the Treatment of Internal Cancer

This course will consider indications and contraindications for megavoltage therapy in the treatment of malignant disease. Supplemental therapy will be discussed. Treatment programs and end results at the University of California at Los Angeles will be presented.

COURSE 223

Wednesday

SIMON KRAMER, M.D.
Philadelphia, Pennsylvania

Radiotherapeutic Retreatment: To Do or Not To Do?

The radiologist is commonly confronted with the problems of retreating recurrent malignancies previously irradiated or of treating new lesions in areas previously irradiated.

The discussion will focus on principles to be observed in deciding on appropriate procedures and also on methods to be utilized. Specific problems will be discussed to elucidate the principles involved.

COURSE 224

Wednesday

CHARLES L. MARTIN, M.D.
Dallas, Texas

The Treatment of Pulmonary Metastases with Roentgen-Ray Therapy

Most radiologists take a pessimistic attitude toward the treatment of metastatic pulmonary lesions with irradiation because it is a well known fact that the administration of cancerocidal doses to both lungs can produce a fibrosis with a fatal outcome in a relatively short time. No doubt many are also unfamiliar with an excellent paper published by Evans and Leucutia in 1924 in which it was demonstrated that relatively small doses of roentgen rays caused many pulmonary metastases to regress without the production of lung damage.

The technique, which must be carefully carried out to be effective, requires only conventional equipment and a series of slides will be shown to illustrate results and technical pitfalls. This method is not curative but may give the acutely ill patient one to three years of comfortable living.

COURSE 225

Wednesday

VICTOR A. MARCIAL, M.D.
San Juan, Puerto Rico

Cancer of the Tongue: Its Therapeutic Management

Consideration will be given to the natural history and incidence of cancer of the tongue. Aspects of the problem to be discussed are: (1) factors determining method of treatment (radiotherapy or surgery); (2) factors in choice of type of radiotherapy (interstitial, interstitial plus external, isotopes used in place of radium); (3) different techniques of implantation; (4) dosimetry problems; (5) care of patients during and after implantation; (6) complications and their management; and (7) management of lymph node involvement.

COURSE 301

Thursday

ROBERT P. BARDEN, M.D.
Philadelphia, Pennsylvania

Interpretation of Radiologic Signs of Abnormal Pulmonary Function

Knowledge of some aspects of abnormal pulmonary physiology helps the clinical radiologist in two ways: he may diagnose many chest diseases more completely, and he may be able to evaluate the effect of disturbed pulmonary function on other systemic diseases.

This course will show examples of roentgenologic examinations which provide help in evaluating disturbances in ventilation, diffusion and arterial oxygenation.

COURSE 302

Thursday

THEODORE F. HILBISH, M.D.
Bethesda, Maryland
FREDERIC C. BARTTER, M.D.
Bethesda, Maryland

Roentgen Findings in Diseases with Abnormal Calcium Deposition

This course will present the roentgen manifestations of diseases associated with abnormal calcium deposition including such conditions as calcinosis universalis, myositis ossificans, hypoparathyroidism, collagen vascular diseases, etc.

The purpose will be to record the interesting and, at times, unusual roentgen findings associated with abnormal calcifications in the tissues. No attempt will be made to cover every entity causing abnormal calcification but the more interesting and informative conditions will be included. A brief discussion of the differential diagnosis will be given in each instance with the intent of affording the radiologist a quick source of identification as to etiology.

COURSE 303

Thursday

GERALD M. MCDONNEL, M.D.
Los Angeles, California

The Radiation Hazard

The trend in evaluation of the radiation hazard has been to the extreme of conservatism. Often the evaluation is performed by nonmedical persons and, if done within the medical profession, often not by the radiologist. Factual data relating natural radia-

tion hazards of the environment to man-made contamination of the biosphere (fall-out) will be given. Comparison of these values to the radiation dosage obtained in diagnostic radiographic procedures will be made. The implication of these data to the population at large and the responsibility of the radiologist in evaluating the information will be discussed.

COURSE 305

Thursday

EARL BARTH, M.D. (MODERATOR)
Chicago, Illinois
WILBUR BAILEY, M.D.
Los Angeles, California
WALLACE D. BUCHANAN, M.D.
South Bend, Indiana
ARTHUR J. PRESENT, M.D.
Tucson, Arizona
T. J. WACHOWSKI, M.D.
Wheaton, Illinois

Current Opinions from the American College of Radiology on Principles of Ethics and Economics

This course will cover the principles of medical ethics as they apply to radiologic practice in hospitals, offices and clinics. Many facts and figures compiled by the American College of Radiology will be presented.

It will be the purpose of this course to point out how the College can be most useful to its members and thus ultimately assist in developing the best possible radiologic practices. The course should be of special interest to the younger men.

Course 305 will start at 1:30 P.M. and will be held in Napoleon Room No. 3. No tickets will be required for this course.

COURSE 306
and
COURSE 406

Thursday and Friday

WILLIAM H. SHEHADI, M.D.
Port Chester, New York

Radiologic Study of the Biliary Tract

Although some pathologic conditions involve primarily the liver and others the extra hepatic biliary tract, radiologically as well as clinically, the biliary tract should be considered one unit. The anatomy, physiology and pathology of the biliary tract will be briefly reviewed, with special emphasis on a study of liver function tests and jaundice, bringing out the role they play in biliary tract visualization.

The course will encompass the technique of liver examination, liver function tests and jaundice, and a study of liver diseases which are radiologically significant.

Also the techniques of gallbladder and bile duct visualization, the relative value of the oral and intravenous methods, and an evaluation of contrast media used in biliary tract visualization will be covered. The commonly as well as the infrequently encountered diseases of the gallbladder and ducts and the so-called postcholecystectomy syndrome will be discussed from a radiologic point of view.

COURSE 308
and
COURSE 408

Thursday and Friday

BERNARD S. EPSTEIN, M.D.
New Hyde Park, New York

Myelographic Changes Associated with Spinal Osteophytosis

Course 308. The instructor's technique for myelographic examination and the normal radiographic anatomy of the spinal canal will be presented briefly. Variations in the architecture of the vertebrae affecting the available space in the spinal canal will be considered. The clinical and radiologic aspects of spondylosis deformans and concomitant degenerative changes in the intervertebral disks will be reviewed. The consequences of intraluminal protrusion of spurs and ridges into the spinal canal and the intervertebral foramina will be discussed.

The Radiographic Investigation of the Basilar Cisterns

Course 408. The normal anatomy of the basilar cisterns and their pneumoencephalographic demonstration will be reviewed, including mention of the diagnostic aid afforded by laminagraphy and positive contrast examinations. The changes produced by neoplastic lesions impinging upon the basilar cisterns will be discussed, correlating the cisternal abnormalities with those of the adjacent brain and cranium. The effects of various inflammatory changes also will be considered.

COURSE 309
and
COURSE 409

Thursday and Friday

RICHARD G. LESTER, M.D.
Richmond, Virginia

Angiocardiography in the Diagnosis of Operable Heart Disease

The place of angiocardiography and aortography in the diagnosis of many congenital cardiac lesions

and certain acquired diseases is now well established. In these sessions emphasis will be placed on those conditions that are amenable to surgical correction or amelioration. Correlation with postmortem anatomy will be provided in large part by comparison of angiocardiograms and specimens of individual patients.

Introduction to Angiocardiography

Course 309. This session will begin with a discussion of indications for angiocardiography. Methods and techniques will be presented. The equipment necessary for both forward and selective examinations will be noted and the relative merits of rapid-film units and cineradiographic apparatus discussed.

Methods for the analysis of angiocardiographic examinations will follow. Criteria for the diagnosis of the more common lesions will be given. Data of importance to the surgeon will be emphasized.

Advanced Angiocardiography

Course 409. A more detailed analysis of certain of the findings in lesions discussed in the previous session will be provided. In addition, there will be consideration of other lesions of relative rarity but of surgical significance. The place of angiocardiography as a research method will be touched upon.

Participants are invited to show and discuss problem cases during the second session.

COURSE 311
and
COURSE 411

Thursday and Friday

GEORGE T. WOHL, M.D.
Philadelphia, Pennsylvania
JOHN KIRKPATRICK, M.D.
Philadelphia, Pennsylvania

The Role of the Radiologist in Bone Tumor Diagnosis

There have been many recent advances in our knowledge of bone lesions. These make possible a more accurate roentgen diagnosis of many benign and malignant bone tumors. In this course a practical method of analyzing bone lesions useful to the radiologist will be presented.

A simplified pathologic correlation will be discussed. Many cases will be presented drawn from the files of the Philadelphia Orthopedic-Radiologic-Pathologic Conference. In the first session, the classification of bone tumors will be presented. The method of analyzing bone lesions will be reviewed. The various benign bone tumors will be presented for discussion. The second session will cover malignant bone tumors and lesions simulating bone tumors.

COURSE 312
and
COURSE 412

Thursday and Friday

JOHN A. CAMPBELL, M.D.
Indianapolis Indiana
E. C. KLATTE, M.D.
Indianapolis, Indiana

Cineradiography

That cineradiography is a useful research method and teaching aid has been established. It has also received wide acceptance as a practical clinical method in the cardiovascular, genitourinary, and gastrointestinal fields and is gaining popularity in orthopedic, neurologic, and certain other applications. Some type of image intensification must be utilized to permit an acceptable radiation dose to the patient. This may result in a reduction in desirable image detail and limitation in field size.

The diagnostic yields obtained with currently available equipment may be enhanced by a proper understanding of the inherent limitations of the method, the controllable technical factors, and the requirements for successful clinical adaptations. This course will present the technical and clinical considerations in cineradiography as outlined below.

Course 312. *Part I—Technical Considerations:* 1. Historical. 2. Current types of cine systems. 3. Roentgen-ray tube requirements. 4. Table adaptations. 5. Optical systems. 6. Cameras. 7. Image resolution, brightness and contrast requirements. 8. Exposure factors. 9. Kinescopic versus direct recording. 10. Film characteristics. 11. Processing. 12. Film projection and viewing. 13. Filing. 14. Radiation dose. 15. Control of image quality.

Course 412. *Part II—Clinical Considerations:* 1. Advantages and disadvantages of clinical cineradiography. 2. Clinical methodology of (a) cinecardioangiography, (b) cystourethrography, (c) orthopedic applications, (d) neuroroentgenology, (e) gastrointestinal applications, (f) gynecologic applications, (g) respiratory applications. 3. Future developments.

COURSE 313

Thursday

HARRY Z. MELLINS, M.D.
Brooklyn, New York

Nonobstructive Acute Abdominal Disorders

An analytic approach to the roentgenographic diagnosis of the nonobstructive acute abdominal disorders will be presented. Emphasis will be placed upon the plain film findings. The importance of serial or follow-up examinations will be stressed. The course will include inflammatory conditions, trauma, toxic conditions and vascular disorders.

COURSE 321

Thursday

JOHN HALE, Ph.D.
Philadelphia, Pennsylvania

A Diagnostic Physics Laboratory Course for Radiology Residents

Teaching of radiologic physics has tended to overemphasize the physics of therapy and radioactive isotopes. In an attempt to balance this situation, a one week long laboratory course in the physics of diagnostic radiology has been designed for residents at the Hospital of the University of Pennsylvania. This course will be described in some detail. The theoretic background and practical importance of each experiment will be discussed, and typical experimental results will be shown.

The course consists of the following experiments:

Radiography: (1) Basic measurements of the physical characteristics of a radiographic unit. (2) Film response characteristics. (3) Effect of radiographic equipment variables. (4) Effect of darkroom variables. (5) Unsharpness. (6) Perception of the visual image.

Fluoroscopy: (1) Basic physical measurements in fluoroscopy. (2) Contrast and detailed perception of: (a) a conventional fluoroscope, (b) an image intensifier.

COURSE 322

Thursday

M. V. PETERS, M.D.
Toronto, Ontario, Canada

Indications for Radiotherapy during the Course of Hodgkin's Disease

In this presentation the treatment of the new case of Hodgkin's disease according to stage will be reviewed and a suggested plan of management presented based on the findings on admission.

The treatment of recurrent disease is often more puzzling than the management of the newly diagnosed case. For this reason radiotherapy and chemotherapy will be discussed with regard to the more important sites of recurrence.

COURSE 323

Thursday

WALTER T. MURPHY, M.D.
Buffalo, New York

Carcinoma of the Vagina and Female Urethra

Primary carcinomas of the vagina and female urethra occur relatively infrequently. However, they should be of great importance to the radiation therapist because of their impressive radiocurability with resultant preservation of normal pelvic functions. Discussion will cover the clinical-pathologic aspects, the various radiotherapeutic techniques, radiation tolerance of the vulvo-perineal and contiguous pelvic tissues, and end results.

COURSE 324

Thursday

BERNARD ROSWIT, M.D.
Bronx, New York

Cancer Chemotherapy in Radiologic Practice

It is essential that radiologists keep well informed of newer developments in cancer chemotherapy in order to improve their management of patients with advanced disease. This course has, therefore, been planned by a radiologist for radiologists to discuss all available drugs, dosage, indications, clinical benefits, toxicity, complications and, above all, integration of drugs with the radiation treatments. A chemotherapy manual will be provided to participants.

COURSE 401

Friday

EDWARD B. SINGLETON, M.D.
Houston, Texas

Pediatric Chest Abnormalities

1. Technique
2. The normal chest and variations of normal
3. Abnormalities of the thoracic cage
4. Newborn pulmonary complications: a. variations of perinatal distress—hyalin membrane disease, aspiration syndrome and pulmonary hemorrhage; b. pneumonias including aspiration secondary to pharyngeal incoordination and esophageal atresia
5. Congenital anomalies: congenital lobar emphysema, congenital cystic malformation, diaphragmatic abnormalities, pulmonary agenesis and hypoplasia
6. Pulmonary abnormalities acquired in later infancy and childhood: a. types of pneumonia and their complications; b. acquired tracheoesophageal fistula; c. foreign bodies; d. fibrocystic disease; e. bronchiolitis
7. Rare pulmonary conditions: pulmonary alveolar microlithiasis, neoplastic diseases, pulmonary fibrosis, hemosiderosis, cytomegalic inclusion disease

COURSE 402

Friday

WILLIAM T. MESZAROS, M.D.
Chicago, Illinois

Roentgen Cardiology of Adults

1. The normal heart
2. Cardiac mensuration
3. Cardiac chamber enlargement
4. Hypertensive heart disease
5. Rheumatic heart disease
6. Arteriosclerotic heart disease
7. Luetic heart disease
8. Cor pulmonale
9. Miscellaneous disorders: a. thyroid heart disease; b. anemia; c. hemachromatosis; d. Addison's disease; e. dissecting aneurysm; f. scleroderma; g. carcinoid
10. Diseases of the pericardium
11. Congenital heart disease in adults
12. The lungs in heart disease

COURSE 403

Friday

BERTRAM R. GIRDANY, M.D.
Pittsburgh, Pennsylvania

Television Tape Studies of Fluoroscopic Procedures in Pediatric Radiology

The radiology department at the Children's Hospital of Pittsburgh utilizes television tape recording of the fluoroscopic image mediated through a newly developed image orthicon television camera optically coupled to a 9 inch image amplifier. The system permits recording on television tape and simultaneous viewing on the closed circuit television monitor at the same low levels of radiation required for closed circuit television viewing alone.

The television tape recordings are transferred to 16 mm. sound motion picture film for showing on standard projectors with or without sound. These recordings offer unusual opportunities for the study of all aspects of motion radiography.

Standard methods of fluoroscopic examination of infants and children with recordings of normal and abnormal conditions will be shown. Included are cardiospasm, congenital and acquired stenosis of the esophagus, H-type tracheoesophageal fistula without esophageal atresia, pyloric stenosis, palatal speech studies, neurogenic megacolon, malrotation, etc.

COURSE 404

Friday

ROBERT H. FREIBERGER, M.D.
New York, New York

The Roentgen Examination of the Foot

The interpretation of the roentgen examination of the foot with the exceptions of fractures and dislocations is very frequently delegated to the orthopedic surgeon. The course is designed to give the radiologist a basic knowledge of tarsal and forefoot alignment.

The following points will be covered:

1. Technique of roentgen-ray examination
2. An explanation of the terms varus and valgus and how they apply to the foot and ankle. Why the changes in tarsal alignment, primarily of the talus and calcaneus, can be expressed in terms of varus and valgus. A minimal amount of anatomy will be included
3. The normal foot
4. Specific foot deformities such as flatfoot, metatarsus adductus, metatarsus varus, talipes equinovarus, congenital plantar flexion of the talus, calcaneus and calcaneo-cavus
5. Recognition of tarsal coalitions

The course is designed to correlate physical and roentgen examination.

COURSE 407

Friday

LEO S. FIGIEL, M.D.
Detroit, Michigan

The Colon: Roentgenographic Considerations

The material presented in this course will establish evidences of a relationship between the colonic polyp and colonic cancer. The detection, significance and follow-up studies of selected case material dealing with the colonic polyp will be presented.

The incidence of error in the detection of colonic cancer will be discussed and measures to prevent such errors will be stressed.

The ileocecal region and the appendix will be covered in detail. The remainder of the course will deal with the barium enema examination in various obstructive conditions other than carcinoma, *e.g.*, volvulus, colo-colic intussusception and others. The more unusual entities in which a specific diagnosis is frequently possible such as endometriosis, pneumatosis coli, etc., will also be considered.

COURSE 410

Friday

KURT AMPLATZ, M.D.
Minneapolis, Minnesota

Techniques of Angiography

This course will deal with the various methods and techniques of angiography and to some extent with the indications and reasons for selecting one procedure over another. Peripheral arteriography, aortography, renal angiography, coronary arteriography, left ventriculography, carotid, vertebral and subclavian angiography will all be discussed. Percutaneous puncture with insertion of catheters of various types will be stressed. The desirability of using some type of pressure injector will be pointed out as well as many other facets of angiographic technique. Questions will be welcomed.

COURSE 421

Friday

JOSE SALA, M.D.
Columbia, Missouri

Treatment of Carcinoma of the Uterine Cervix

This course will be based on the experience at The Ellis Fischel State Cancer Hospital from 1943 to the present. Different techniques will be reviewed, including the use of transvaginal irradiation and curie therapy. Five year results will be given for the various methods.

COURSE 422

Friday

FRANK BATLEY, M.B., ChB., D.M.R.T.
Syracuse, New York

Common Errors in Radiotherapeutic Technique

The following philosophy of radiotherapy will be illustrated. Cobalt therapy cannot replace interstitial implantations for small mouth lesions, yet, gold grains are often preferable to radium needles. This heresy is held with the belief that only those implantations should be performed in which the dose can be calculated or nearly calculated. Large implants are advised as often as commando surgery.

The method of using parallel opposing fields with external irradiation is the easiest and worst technique. Less than three fields should never or hardly ever be used.

Radiosensitivity and radioresistance are now old-fashioned and misleading concepts, but the terms radiocurable and radio-incurable are not. One should know the limitations—or at least the limitations of the patient's tissues.

Illustrations will include reminiscences of the Holt Radium Institute in Manchester, consisting of the commencement of the "Find Point A Game," the use and abuse of the Paterson-Parker rules demonstrated by an electric toaster, the devaluation of the British roentgen, and the experiences of some early days with the first or second cobalt unit in Canada—2 half lives later.

COURSE 423

Friday

WALTER T. MURPHY, M.D.
Buffalo, New York

Complications of Head and Neck Irradiation

Radiation therapy of head and neck tissues is indicated for a great variety of primary and metastatic neoplasms. Sometimes such treatment precedes or follows surgical or chemotherapeutic procedures. Risks of cancerocidal irradiation are always real even with modern techniques. Avoidance of complications is made easier by an understanding of what can happen to patients irradiated for different clinical entities and with different radiotherapeutic techniques.

COURSE 424

Friday

JAMES J. NICKSON, M.D.
New York, New York

Applied Radiation Biology

In this course the fundamentals of radiation biology with particular reference to the clinical situation will be discussed. Consideration will be given to the opportunities that exist for improvement of the results of radiation therapy, and also to the opportunities that exist for advancing our knowledge in radiation biology by appropriately designed observations and trial studies in patients.

COURSE 425

Friday

FERNANDO G. BLOEDORN, M.D.
Baltimore, Maryland

The Combined Use of Radiotherapy and Surgery in Treatment of Cancers of the Lung and Esophagus

A program of combined radiotherapy and surgery for lung and esophageal carcinomata was established at the University of Maryland Hospitals in 1956.

This course will consider the basis for such an effort, the reasons for failure to cure these lesions, the philosophy and techniques of treatment, the complications of the method, and an analysis of results.

CONDENSED SCHEDULE OF COURSES

TUESDAY **3:00 to 4:30 P.M.** **September 26, 1961**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine Physics and Radiobiology	
101—Finby	Male Cystourethrography	**121—del Regato**	El Papel de la Radioterapia en el Tratamiento del Cancer (In Spanish)
102—Hope	Roentgen Facts and Fallacies Regarding the Normal and Abnormal Genito-urinary Tract of Infants and Children	**122—Sanger**	Diagnostic Uses of Radioisotopes (Other Than for Thyroid Disease)
103—Morgan	Screen Intensification and Cineradiography	**123—Williams**	The r, rad and rem in Specification of Dose
104—Rubin	The Development of a Dynamic Classification of Bone Dysplasias	**124—D'Angio**	Radiation Therapy in the Management of Children with Tumors
105—Tuddenham	New Concepts in Radiographic and Interpretative Techniques		
106—Etter	Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses		
107—Weens Gay	Roentgenology of the Pulmonary Circulation—Diagnosis of Certain Physiologic Changes		
108—Levin	The Fundamentals of Bone Disease		
109—Macmillan	Sinuses		
110—Nelson	Bronchography		
111—Wittenborg	Pelvis and Hips in Infancy and Early Childhood		
112—Egan	Over Five Years' Experience with Mammography		
113—Marshak	The Small Intestine		

CONDENSED SCHEDULE OF COURSES

WEDNESDAY **3:00 to 4:30 P.M.** **September 27, 1961**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
201—Fischer Dodd Debbas	Lymphography	**221—Sheline**	The Use of Radioisotopes in the Diagnosis and Treatment of Thyroid Disease
202—Hope	Mediastinal Mass Lesions in Infants and Children	**222—Stein**	The Use of Megavoltage Therapy in the Treatment of Internal Cancer
203—Nice	Roentgenographic Manifestations of Collagen Diseases	**223—Kramer**	Radiotherapeutic Retreatment: To Do or Not To Do?
204—Jacobson	Malignant Bone Tumors—Primary and Secondary	**224—Martin**	The Treatment of Pulmonary Metastases with Roentgen-ray Therapy
205—Dotter (Same as 405)	The Mechanism and Practical Management of Cardiovascular Emergencies Likely To Arise in a Radiology Department	**225—Marcial**	Cancer of the Tongue: Its Therapeutic Management
206—Etter	Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses		
207—Felson	Some Fundamentals in Chest Roentgen Interpretation		
208—Levin	The Fundamentals of Bone Disease		
209—Macmillan	Mastoids, Lateral Neck and Larynx		
210—Nelson	Bronchography		
211—Schatzki	Differential Diagnosis of Lesions at the Lower End of the Esophagus		
212—Eyler Ormond	The Acute Abdomen		

CONDENSED SCHEDULE OF COURSES

THURSDAY **3:00 to 4:30 P.M. (except Course 305)** **September 28, 1961**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
301—Barden	Interpretation of Radiologic Signs of Abnormal Pulmonary Function	321—Hale	A Diagnostic Physics Laboratory Course for Radiology Residents
302—Hilbish Bartter	Roentgen Findings in Diseases with Abnormal Calcium Deposition	322—Peters	Indications for Radiotherapy during the Course of Hodgkin's Disease
303—McDonnel	The Radiation Hazard	323—Murphy	Carcinoma of the Vagina and Female Urethra
304—Jacobson	The Vaterian Segment: Normal and Abnormal	324—Roswit	Cancer Chemotherapy in Radiologic Practice
305*—Barth Bailey Buchanan Present Wachowski	Current Opinions from the American College of Radiology on Principles of Ethics and Economics		
306—Shehadi	Radiologic Study of the Biliary Tract		
307—Felson	Some Fundamentals in Chest Roentgen Interpretation		
308—Epstein	Myelographic Changes Associated with Spinal Osteophytosis		
309—Lester	Introduction to Angiocardiography		
310—Marshak	The Small Intestine and Colon		
311—Wohl Kirkpatrick	The Role of the Radiologist in Bone Tumor Diagnosis		
312—Campbell Klatte	Cineradiography. Part I.		
313—Mellins	Nonobstructive Acute Abdominal Disorders		

*** Course 305 will start at 1:30 P.M. and will be held in Napoleon Room No. 3.**

CONDENSED SCHEDULE OF COURSES

FRIDAY **1:30 to 3:00 P.M.** **September 29, 1961**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
401—Singleton	Pediatric Chest Abnormalities	**421—Sala**	Treatment of Carcinoma of the Uterine Cervix
402—Meszaros	Roentgen Cardiology of Adults	**422—Batley**	Common Errors in Radiotherapeutic Technique
403—Girdany	Television Tape Studies of Fluoroscopic Procedures in Pediatric Radiology	**423—Murphy**	Complications of Head and Neck Irradiation
404—Freiberger	The Roentgen Examination of the Foot	**424—Nickson**	Applied Radiation Biology
405—Dotter (Same as 205)	The Mechanism and Practical Management of Cardiovascular Emergencies Likely To Arise in a Radiology Department	**425—Bloedorn**	The Combined Use of Radiotherapy and Surgery in Treatment of Cancers of the Lung and Esophagus
406—Shehadi	Radiologic Study of the Biliary Tract		
407—Figiel	The Colon: Roentgenographic Considerations		
408—Epstein	The Radiographic Investigation of the Basilar Cisterns		
409—Lester	Advanced Angiocardiography		
410—Amplatz	Techniques of Angiography		
411—Wohl Kirkpatrick	The Role of the Radiologist in Bone Tumor Diagnosis		
412—Campbell Klatte	Cineradiography. Part II.		

SECTION ON INSTRUCTION
ORDER SHEET

It is important to register for the Instruction Courses as early as possible since the number admitted to each course will be limited. *It is also very important that first, second and third choices be listed for each period.* All orders for tickets will be filled according to the postmark on the envelopes.

Non-members of the American Roentgen Ray Society, except full-time graduate students in Radiology, will pay $2.00 for each course-period or a maximum of $8.00 for 4 courses. *Non-members' fees must accompany this order sheet and will not be returned unless cancellation is received before September 20, 1961.*

Tickets for courses will be mailed to each registrant whose order reaches the office of the Director on or before September 12, 1961. They will be sent to the address below unless registrant directs otherwise.

Please fill out the following (type or print):

Check ☐ Member ☐ Guest ☐ Graduate Student in radiology at:

.....
Last Name First Name or Initials

.....
Street Address

....
City Zone State

Those whose advance registration reaches the office of the Director after September 12, 1961, may obtain their tickets at the Registration Desk of the Section on Instruction on Monday afternoon, September 25, 1961 and thereafter. At this time tickets may also be obtained for courses not sold out.

For convenience in selecting your courses consult the condensed schedule of courses.

BE SURE TO FILL OUT SECOND AND THIRD CHOICES FOR EACH PERIOD

Period	First Choice		Second Choice		Third Choice	
	Course No.	Instructor	Course No.	Instructor	Course No.	Instructor
Tuesday						
Wednesday						
Thursday						
Friday						

Previous to September 12, 1961, send this order sheet to:

DR. HAROLD O. PETERSON, 1995 WEST COUNTY ROAD "B," ST. PAUL 13, MINNESOTA.

After September 12, 1961, mail to:

DR. HAROLD O. PETERSON, American Roentgen Ray Society, Deauville Hotel, Miami Beach, Florida.

BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

The Atom and the Energy Revolution. By Norman Lansdell. Cloth. Price, $6.00. Pp. 200, with 32 illustrations. Philosophical Library, Inc., 15 East 40th Street, New York, N. Y., 1958.

This very readable book is addressed to the "general reader" who wishes to learn more about the social and political impact of the great atomic energy revolution. It should prove of interest to the men in business and industry, who must adapt themselves to the new economy, as well as to the specialist in related fields of inquiry. After a discussion of world resources of fuel and of expected world demands for increasing amounts of energy, the author describes various new sources of energy, notably, atomic energy. Fission and fusion energy and their releases are discussed in detail, followed by a description of the natural sources of materials for atomic energy developments. Interesting ideas are presented on radiation risks and insurance against them, on the influence of atomic energy development upon other industrial techniques, on the influence of atomic energy on the balance of world trade and finally on the relation of private industry to the state in respect to atomic energy. The wide scope of the impact on world affairs from the development of atomic energy is impressively presented. Unfortunately, the book has no index, which, in a work of this type, written for the general reader, would have been especially useful. A three-page glossary of scientific terms partially makes up for this omission.

Otto Glasser, Ph.D.

BOOKS RECEIVED

The Spinal Cord; Basic Aspects and Surgical Considerations. By George Austin, Professor and Head, Division of Neurosurgery, University of Oregon Medical School; Chief Consultant in Neurosurgery, Veterans Administration Hospital, Portland, Oregon; Formerly, Assistant Professor of Neurosurgery, University of Pennsylvania. Cloth. Price, $26.50. Pp. 532, with 363 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

Protection Against Radiation; A Practical Handbook. By John D. Abbatt, M.B., Ch.B., D.M.R.; J.R.A. Lakey, Ph.D., B.Sc., A.Inst. P.; and D. J. Mathias. Cloth. Price, $6.50. Pp. 235, with 44 illustrations and 35 tables. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

Cancer of the Nasopharynx: Its Natural History and Treatment. By M. Lederman, M.B., D.M.R., F.F.R., Deputy Director, Radiotherapy Department, Royal Marsden Hospital; Consulting Radiotherapist, The Royal National Throat, Nose and Ear Hospital, The Metropolitan Ear, Nose and Throat Hospital, Moorfields Eye Hospital, The Royal Eye Hospital, The Chelsea Hospital for Women, London, England. Cloth. Price, $6.75. Pp. 117, with 53 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

Problems of Infection, Immunity and Allergy in Acute Radiation Diseases. From the Russian by N. N. Klemparskaya; O. G. Alekseyeva; R. V. Petrov; and V. F. Sosova. Translated by Lydia Venters, Argonne National Laboratory, Lemont, Illinois, Cloth. Price, $7.50. Pp. 165, with 34 illustrations and 29 tables. Pergamon Press, Inc., 122 East 55th Street, New York 22, N. Y., 1961.

Le Neoplasie Maligne della Tiroide; Diagnosi e Terapia. By Franco Chiariotti. Paper. Pp. 187, with many illustrations and tables. Minerva Medica, 1961.

Annual Review of Medicine. Volume 12. David A. Rytand, Editor, Stanford University School of Medicine; and William P. Creger, Associate Editor, Stanford University School of Medicine. Cloth. Price, $7.00. Pp. 455, with few illustrations and tables. Annual Reviews, Inc., Palo Alto, California, 1961.

IXth International Congress of Radiology. Volume I. Edited by Prof. B. Rajewsky, Dr. phil. nat., Dr. med. h. c., Dr. med. h. c., Frankfurt a. M., Präsident des Kongresses. Cloth. Pp. 800, with numerous illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961. In the U. S. A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

IXth International Congress of Radiology. Volume II. Edited by Prof. B. Rajewsky, Dr. med. h. c., Dr. med. h. c., Frankfurt a. M., Präsident des Kongresses. Cloth. Pp. 825, with numer-

ous illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961. In the U. S. A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Acta Radiologica Supplementum 196. Mammary Carcinoma; the Biologic Character of Mammary Carcinoma Studied in 517 Cases by a New Form of Malignancy Grading. By K. A. Hultborn and Bo Törnberg. Paper. Price, Sw.Kr. 35:-. Pp. 143, with 74 illustrations and 140 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplementum 197. The Mitotic and Radioprotective Effect of Cysteine and Lysine in Rat. By Lars R. Holsti. Paper. Price, Sw. Kr. 30:-. Pp. 99, with 17 illustrations and 18 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplementum 198. The Isometric Relaxation Phase of the Left Ventricle; an Electrokymographic Study. By Osborne Bartley. Paper. Price, Sw. Kr. 35:-. Pp. 116, with 49 illustrations and 14 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplementum 199. Vaso-Seminal Vesiculography in Hypertrophy and Carcinoma of the Prostate with Special Reference to the Ejaculatory Ducts; a Clinical and Experimental Roentgenological Study. By Gunnar Willer Vestby. Paper. Price, Sw. Kr. 35:-. Pp. 194, with 119 illustrations and 17 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplementum 200. Contrast Examination of the Cardiovascular System during Increased Intrabronchial Pressure. By Björn Nordenström. Paper. Price, Sw. Kr. 30:-. Pp. 110, with 51 illustrations and 1 table. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1960.

Acta Radiologica Supplement No. 201. Risa Encephalography and Conventional Neuroradiologic Methods; a Comparative Study. By Giovanni Di Chiro. Paper. Price, Sw. Kr. 35:-. Pp. 102, with 38 illustrations, 60 sketches and 3 diagrams. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1961.

Therapeutic Dose Distributions with High-Energy Radiation. Survey of Existing Data and Report of a Panel on Physical Data for Dose Distributions with High-Energy Radiation Held at Vienna, 7–11 November 1960. Paper. Pp. 72. International Atomic Energy Agency, Kaerntnerring 11, Vienna 1, Austria, 1961.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Skeletal System

Hunt, James Cleon, and Pugh, David G. Skeletal lesions in neurofibromatosis. *Radiology*, Jan., 1961, *76*, 1–20 (Address: J. C. Hunt, Mayo Clinic, Rochester, Minn.)

Neurofibromatosis (Recklinghausen's disease) is a congenital and familial disease presenting abnormalities of the skin, nervous system, bones and soft tissues. While the neurectodermal tissues have been accepted as being the tissues more commonly involved, recent studies have shown that mesodermal dysplasia is also a basic component, accounting for most of the skeletal defects found in this disease.

A total of 192 cases in which a positive diagnosis of neurofibromatosis was made during a ten year period from 1948 through 1957 at the Mayo Clinic was studied for skeletal defects. In 97 (51 per cent) some skeletal abnormality was demonstrated roentgenographically. These changes, when analyzed, classified and tabulated, revealed that skeletal defects of a type considered to be characteristic were present in 74 cases (39 per cent) and should suggest the possibility of this disease to the radiologist.

These defects include: severe angular scoliosis with dysplasia of vertebral bodies (14 cases); defects of the posterosuperior wall of the orbit (13 cases); disorders of bone growth associated with elephantoid hypertrophy of overlying soft tissue (18 cases); congenital bowing and pseudoarthrosis (4 cases); erosive defects of bone from contiguous neurogenic tumors (36 cases); and intrathoracic meningocele (1 unproved case).

Other skelecal abnormalities, not characteristic but possibly representing mesodermal dysplasia, consisted of congenital anomalies of many types and a mild scoliosis. Thirty-three examples were found of the former and 14 of the latter. Intraosseous cystic lesions, described by some writers as a characteristic manifestation of neurofibromatosis, were not thought by the authors to fall in this classification. No instances of this lesion were found in this series of cases. Osteomalacia, considered by some to be an incidental finding in neurofibromatosis, was also not found.

The incidence of neurofibromatosis among admissions to the Mayo Clinic during the ten year period was 1 in 3,100, and there were 9 cases (5 per cent) in which sarcomatous degeneration occurred.—*D. N. Dysart, M.D.*

Retz, Louis D., Jr. Primary liposarcoma of bone: report of a case and review of the literature. *J. Bone & Joint Surg.*, Jan., 1961, *43-A*, 123–129. (From: Department of Surgery, State University of New York, Upstate Medical Center, and the Surgical Service, Veterans Administration Hospital, Syracuse, N. Y.)

A case is reported of a liposarcoma confined to the medullary cavity of the tibia. A forty year old male had a single well circumscribed central lytic defect in the upper metaphysis of the tibia measuring 4 by 5 cm. in size. The epiphyseal plate was intact. There was no periosteal reaction or cortical erosion. There was loss of the normal trabecular pattern and a "ground glass" appearance.

The lesion was curetted and packed with bone but four months later there was extension down the shaft. A midthigh amputation was done and two years later there was no evidence of recurrence or metastases.

Pathologic examination showed no tumor outside the bone and no cortical breakthrough. The tumor was well differentiated, with areas of typical nonossifying fibroma associated with sarcoma and an abundance of intracellular lipids. There were spindle shaped foamy lipoblasts and giant vacuolated sarcomatous cells. Considerable lymphocytic infiltration was seen but no necrosis.

Liposarcomas are found most frequently in the thigh, popliteal space, gluteal region and retroperitoneal space. Only 7 cases of intramedullary lipoma and 14 cases of liposarcoma of bone have been reported. None of the latter were confined solely to the medullary cavity, as in the present case. The more differentiated liposarcomas are slow growing and usually do not metastasize, but they may recur if they are not adequately excised. The less differentiated tumors have a poor prognosis and metastasize through the blood stream, commonly to the lungs, other bones and the brain.—*Martha Mottram, M.D.*

Scaglietti, O., and Stringa, G. Myxoma of bone in childhood. *J. Bone & Joint Surg.*, Jan., 1961, *43-A*, 67–80. (From: Istituto Ortopedico Toscano dell'Università di Firenze, Florence, Italy.)

This rare bone tumor closely resembles chondromyxoid fibroma but always shows recurrence after simple curettage; therefore, initial resection is indicated when the diagnosis is established.

Two new cases are presented as well as 3 previously reported, all of which developed during the first decade. There was rapid development of pain, swelling and limitation of motion.

The lesions were visualized roentgenographically as irregularly rounded, lobulated, eccentric radiolucent defects in the metaphyses and showed a marked increase in the thickness of the cortical bone and diffuse sclerosis of the underlying spongiosa. Grossly, they resembled gelatinous cysts with septa. The tumor appears to arise in the subperiosteal extracortical region and penetrates the spongiosa. Each lobule of the tumor contained elongated stellate or triangular cells with a margin of closely packed round cells and foreign body giant cells. The intercellular substance had a uniform thin fibrillar appearance with metachromatic staining. An older myxoma may

develop fibromatous elements, so it may be termed fibromyxoma.

Although some might classify myxomas as juvenile forms of chondromyxoid fibroma because they appear so similar, it is urged that myxomas be considered in a separate category since they occur before the age of ten and are characterized by marked pain and swelling with rapid progression, marked periosteal reaction seen on roentgenograms, predominance of myxoid tissue and a positive reaction for metachromasia in the ground substance, and invariable recurrence after simple curettage but complete recovery after resection.—*Martha Mottram, M.D.*

Schajowicz, Fritz. Giant-cell tumors of bone (osteoclastoma); a pathological and histochemical study. *J. Bone & Joint Surg.*, Jan., 1961, *43-A*, 1–29. (From: Center of Osteoarticular Pathology and the Department of Orthopaedic Surgery and Traumatology, Italian Hospital, Buenos Aires, Argentina.)

This is a review of 85 giant cell tumors with 41 complete specimens available, so that histochemical studies and tissue cultures could be done. Multiple histochemical studies were carried out in giant cell tumors as well as in other bone and soft tissue lesions which contained giant cells. For details as to types of enzymes, carbohydrates and lipids tested, the reader is referred to the original paper.

The author believes from his pathologic studies that the giant cell is of mesenchymal origin and is formed by agglutination and conglomeration of the mononuclear cell as well as by repeated division of the stromal cell nuclei by amitosis or nuclear segmentation. He feels that the giant cell tumor is a neoplastic derivation of the osteoclast.

Giant cells have an abundance of acid phosphatase, betaglucuronidase, succinic-dehydrogenase and no alkaline phosphatase. There are also complex lipids, probably glyco- or phospholipids. The stromal cells of giant cell tumors generally show no enzymatic reactions, nor do they possess glycogen, mucopolysaccharides or glycolipids in significant amounts.

The histochemical findings are identical in the multinucleated giant cells of giant cell tumors and the so-called variants and in the normal chondroclastic and osteoclastic giant cells.

The author concludes that histochemical study is of no diagnostic value in differentiating giant cell tumor from its variants or in defining prognosis. In the exceptional case with rapid evolution and recurrence, differentiation from osteolytic osteogenic sarcoma can be made easily by the presence of intense alkaline phosphatase activity in the latter. The histologic, clinical and roentgenographic findings could not be correlated sufficiently to grade the giant cell tumors.

Radical resection of the tumor with bone grafts is recommended because of an increasing number of cases of benign or agressive giant cell tumors which have produced metastases with identical histologic patterns and which showed progressive growth, recurred or transformed into sarcoma.—*Martha Mottram, M.D.*

Bose, K. S. Observations on changes of pattern in chronic pyogenic osteomyelitis following inadequate administration of penicillin: their similarity with other bone diseases. *J. Indian M. A.*, Apr., 1959, *32*, 271–273. (From: Fracture and Orthopaedic Department, Medical College, Calcutta, India.)

This is a report on 7 cases of chronic pyogenic osteomyelitis which had been inadequately treated with antibiotics and which presented atypical features simulating various lesions of bone, but excluding malignancy. These cases were investigated from clinical, roentgenologic and histologic aspects, with an attempt to categorize the lesions insofar as possible. A definitive diagnosis on the basis of the clinical and roentgen findings is often difficult and may depend on an adequate biopsy specimen and evaluation of the clinical course over a period of time.

It was found that inadequate antibiotic administration in the acute stage of pyogenic osteomyelitis resulted in unchecked virulence of the organism, more marked bony necrosis and rarefied areas which roentgenographically simulated bone diseases such as hemangioma, fibrocystic disease and Gaucher's disease. Moreover, in other case, new bone formation was seen to be more marked, simulating sclerosing bone diseases such as osteoid osteoma, Paget's disease, infantile cortical hyperostosis, and Engelman's disease.—*Raymond V. Hussey, Captain, USAF MC*

Bose, K. S. Atypical osteomyelitis following inadequate antibiotic therapy: its similarity with fibrous dysplasia. *J. Indian M. A.*, Dec., 1959, *33*, 464–468. (From: Department of Surgery, Medical College Hospitals, Calcutta, India.)

Four cases of chronic pyogenic osteomyelitis following inadequate penicillin treatment are presented with an analysis of the clinical features, roentgen findings, histology, treatment and results.

The roentgen changes included minimal bone necrosis with absence of sequestra and little new bone formation. Also, there was a slowly developing cystic osteoporosis involving the diaphysis with expansion of the shaft and an altered trabecular pattern usually associated with fibrous dysplasia.

The striking feature of this investigation was that 2 of the 4 cases showed typical histologic and roentgen features of fibrous dysplasia but positive biopsy cultures revealed organisms of *Streptococcus viridans* and *Bacillus fecalis alcaligenes*.

The author concludes that infection may well be one factor in the etiology and pathogenesis of fibrous dysplasia.—*Raymond V. Hussey, Captain, USAF MC*

Sutro, C. J., Ehrlich, D. E., and Witten, M. Low back disorders; values of radiographs of the dorsal vertebrae in evaluating the etiology of lumbosacral derangements. *Bull. Hosp. Joint Dis.*, Apr., 1960, *21*, 42–49. (From: Brooklyn Outpatient clinic, U. S. Veterans Administration Hospital, Brooklyn, N. Y.)

Patients with severe pains in the lumbar area or in the sacroiliac region may, on roentgen examination, present minimal or negative findings in the soft or osseous parts of the lumbar vertebrae or of the pelvic bones.

It should be remembered that the lower dorsal nerves innervate the skin in the lumbar, sacroiliac and femoral trochanteric regions. Thus, lesions situated in the lower dorsal vertebrae or in their contiguous soft parts may cause referred pains in the lumbar or pelvic regions.

The authors present 3 cases of such referred pain illustrating the shortcomings of segmental roentgen examination and emphasizing the necessity of including roentgen examination of the dorsal vertebrae in addition to that of the lumbar and pelvic bones in cases of low back disorders.—*Charles H. Helmen, M.D.*

Vyhnánek, Luboš, Lavička, Josef, and Blahoš, Jaroslav. Roentgenological findings in gout. *Radiol. Clin.*, July, 1960, *29*, 256–264. (From: The Radiological Clinic of the Charles-University in Prague, and the Clinic of Internal Medicine in Pilsen, Czechoslovakia.)

Six per cent of the patients with chronic inflammatory rheumatism were found to be suffering from gout. The authors describe the roentgen changes in gout based on their experience with 44 patients.

These changes are divided into three groups: in Group I (20 patients) the findings were negative or nonspecific, in Group II (11 patients) there was localized osteoporosis of the joint and subchondral cystoid areas without damage to the bony cortex; and in Group III (7 patients) there were cystoid areas with damage to the bone cortex and destruction of the joint.—*James J. McCort, M.D.*

Blood and Lymph System

Sheehan, R., Hreshchyshyn, M., Lin, R. K., and Lessmann, F. P. The use of lymphography as a diagnostic method. *Radiology*, Jan., 1961, *76*, 47–53. (Address: R. Sheehan, Roswell Park Memorial Institute, Buffalo 3, N. Y.)

The lymphatic system has been visualized roentgenographically by a number of investigators using the injection of opaque substances, but the first real clinical application was not made until 1952.

The authors report their experiences with lymphangiography undertaken as a diagnostic method in 40 patients with suspected disease of the pelvic or retroperitoneal lymph nodes. The lymphatics were first outlined with direct sky blue or Evans blue dye injected subcutaneously into the web of the foot between the first and second phalanges. A cut down was then made on the dorsum of the foot; the lymph vessel was located; and the contrast medium was injected. In 7 cases, a water soluble medium was used, with only 3 satisfactory results. An oily medium was employed in 31 studies with satisfactory diagnostic results in 27 of these. Ethiodol, which contains 37 per cent iodine in organic combination with the ethyl esters of the fatty acid of poppyseed oil, seemed to produce the best results.

In general, the lymph nodes were visualized up to the level of L-2. Complications were minimal, with gradual absorption of the contrast medium. The procedure seemed to have greater diagnostic value in lymphomas than in metastatic lymph node involvement. Small metastatic lesions were not detectable. The lymph nodes completely filled with tumor did not take up the dye, but displacement of the neighboring channels was sometimes shown. Incomplete filling or inflammatory changes occasionally caused defects which were difficult to interpret.

Venography or intravenous pyelography can visualize pathologic lesions in many cases, while lymphography can be of value in cases where lymphogenous involvement cannot be determined by conventional methods. Lymphography can also be used to explore the normal lymphatic system and to evaluate changes following chemotherapy. The procedure is time consuming and experience is needed for correct interpretation of the results.—*James C. Moore, M.D.*

Radiation Therapy

Arneson, A. N., and Williams, Carol F. Long-term follow-up observations in cervical cancer. *Am. J. Obst. & Gynec.*, Oct., 1960, *80*, 775-790. (From: Department of Obstetrics and Gynecology, and the Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Mo.)

The authors report a statistical study of their long term follow-up observations in cervical cancer. A consecutive series of 394 patients seen between the years 1935 and 1954 was reviewed. Of this group, there were 374 patients with invasive cancers who received treatment: 32 per cent were Stage I, 48.5

per cent Stage II, 16.5 per cent Stage III, and 3 per cent Stage IV. The five year survival for Stage I cases was 78.3 per cent, for Stage II 54.1 per cent, and for Stage III 37.1 per cent. There were no Stage IV five year survivals. The five year over-all survival rate was 57.5 per cent. The five year survival values give a realistic view of cure rates. A patient clinically well after five years has little probability of dying from cancer.

A five to twenty year follow-up is also reported for this series of patients. To test the effectiveness of treatment over the years, the series is divided into two groups of approximately equal numbers—those seen before and those after 1949. The relative five year results for these two groups are compared. An increase in survival is not demonstrated in the more recently treated patients, despite the fact that the percentage distribution of early lesions increased. This difference in results is then studied by using cumulative survival rates.

The authors' experience with programs of secondary irradiation has been unfavorable in the treatment of persistent or reappearing cancer. In more than 50 such attempts, only 2 patients have survived an additional five years after a second course of radiation therapy.

The rate of major complications, such as the incidence of fistula formation, has fallen from 11 per cent in the earliest years to 2 per cent in the 1950–1954 period. This improvement is taken as evidence of an advance in treatment technique, despite the lack of increased survival rates for the most recent years.—*Richard L. Jontz, M.D.*

Sotto, Luciano S. J., Graham, John B., and Pickren, John W. Postmortem findings in cancer of the cervix; an analysis of 108 autopsies in the past 5 years. *Am. J. Obst. & Gynec.*, Oct., 1960, *80*, 791–794. (From: Departments of Gynecology and Pathology, Roswell Park Memorial Institute, Buffalo, N. Y.)

From June, 1954 to May, 1959, a total of 108 autopsies were performed at Roswell Park Memorial Institute on patients diagnosed and treated previously for carcinoma of the uterine cervix. Of these patients 85 per cent had been treated during the preceding five years; 75 per cent had received their primary treatment at the Institute; and 90 per cent of the total had received what is now considered to be adequate treatment.

In 23 per cent, the original stage of disease was unknown because of initial treatment elsewhere; 21 per cent were Stage I, 23 per cent Stage II, 21 per cent Stage III, and 12 per cent Stage IV.

Regarding the clinical response to primary treatment, 37 per cent had persistent pelvic disease, 52 per cent recurrent pelvic disease, 9 per cent no pelvic tumor, and 2 per cent unknown.

The leading cause of death was renal failure (37 per cent), manifested by uremia due to obstruction or pyelonephritis.

Of the 93 patients having tumor present at autopsy, in only 15 per cent was the disease limited to the pelvis. In 28 patients there was bladder involvement and in only 12 was the rectum invaded.

Distant metastases were found in 79 patients (85 per cent) with the lung (38 per cent) and liver (37 per cent) being the most common sites; 20 per cent of the cases had bone involvement, mainly in the spine.

Lymph node metastases were found in 61 of the 93 patients having disease; in 6 instances these were localized in the pelvis; in 33 they were extrapelvic; and in 22 they were both pelvic and extrapelvic.—*R. L. Turner, M.D*

Sheline, Glenn E., Goldberg, Minnie B., and Feldman, Robert. Pituitary irradiation for acromegaly. *Radiology*, Jan., 1961, *76*, 70–75. (Address: G. E. Sheline, Department of Radiology, University of California Medical Center, San Francisco 22, Calif.)

This paper presents the experience gained between January, 1942 and March, 1959 at the University of California Hospitals, San Francisco, with radiation therapy for acromegaly in 37 patients. During this time the diagnosis of acromegaly was made in a total of 65 patients. Seven cases were discarded because of inadequate information, 9 were considered quiescent, 2 patients had hypophysectomy and 1 was treated by estrogen therapy alone. Nine other patients were excluded for various reasons.

In this series, both the "interval" and the "single course" techniques were employed. Ten patients received a calculated pituitary dose of 1,000 to 2,500 r. In the other 27 patients, the dose for the initial course of treatment was 2,550 to 6,000 r, usually given in a period of four to eight weeks. Subsequently, several of those given doses between 2,550 and 3,500 r received additional therapy. External roentgen rays (200, 400 or 1,000 kvp.) were used with multiple stationary fields or a rotational technique. Therapy was usually begun with a daily pituitary dose of 50 r or less and gradually increased to an average of about 700 r per week. The daily dose was determined by the patient's tolerance. The results of irradiation were gauged according to the response evident at six weeks, three months, and twelve months following completion of each course of therapy. The follow-up period ranged from one to eighteen years.

Twenty-eight of the 37 patients improved following the initial course of roentgen therapy. In 19 the disease was permanently controlled. The improvement was either incomplete or transient in 9. A dose in excess of 3,500 r resulted in improvement in 78 per cent of the cases, whereas a lower dose brought the disease under control in only 26 per cent; the latter

figure was raised to 63 per cent by retreatment. The disease was considered to be quiescent if headache, progression of visual field impairment, sellar destruction, skeletal growth, soft tissue swelling and other evidence of endocrine hyperactivity ceased. The visual fields became normal in 10 of the 14 patients for whom data are available.

These results seem to confirm the contention that visual field defects in acromegaly do not constitute a contraindication to pituitary irradiation unless they are recent, acute, and rapidly progressive. No significant complication developed in any patient receiving a single course of treatment, even with tumor doses up to 6,000 r.—*A. W. Sommer, M.D.*

Chiariotti, Franco. Alcuni singolari aspetti delle neoplasie tiroidee. (Unusual aspects of thyroid neoplasms.) *Radiol. med.*, Aug., 1960, *46*, 738–756. (From: Ospedale di Circolo di Busto Arsizio, Italy.)

In a broad sense thyroid cancer is characterized by "unusual aspects" from the anatomic, pathologic, clinical, diagnostic and therapeutic standpoints. The observations which are presented are the result of the study of 164 cases of thyroid tumors treated in the hospital by different methods.

(1) *Discrepancies between the histologic characteristics and the clinical course of thyroid tumors.* The author notes the long survival of patients with diffuse visceral and skeletal metastases and observes that tumor types that might be classified as benign cytologically, may be the origin of extensive generalized metastases (benign metastasizing struma, benign metastasizing thyroid adenoma). He surveys the contributions of other investigators and cites pertinent cases studied by him. Most interesting among these is that of a woman thirty-six years of age who presented a diffuse nodular metastatic dissemination in both lungs which had remained stationary for thirteen years with no appreciable complaints. The functional thyroid origin of the metastases was established by scanning following the administration of I^{131}.

(2) *Malignant tumor in aberrant thyroids.* Topographically one may describe aberrant thyroids as median and lateral. The median which are derivations of the thyroglossal duct have been designated as lingual. Because of the frequency of the malignancy in these sites, prompt determination of the nature of the lesion and therapeutic intervention are indicated. It is difficult to differentiate between cervical lymph node metastases and lateral aberrant thyroids.

(3) *Malignant thyroid tumors in the young.* A survey of the cases reported in the literature and of those included in this study apparently points to an increase in the incidence of malignant tumors observed in the first decades of life. The author notes that, in 4 of the 12 cases he studied, the first symptom was the appearance of lateral cervical tumefactions. Juvenile thyroid carcinomata react favorably both in the primary site and in metastases, especially when surgical intervention is combined with internal and external radiation therapy. He reports an average survival of six to twelve years in subjects still alive.

(4) *Goiter and thyroid cancer.* The incidence of thyroid cancer in previously existent goiter, as reported by the author and by other contributors quoted by him, is between 20 per cent and 50 per cent.

(5) *The relationship of previous irradiation of the neck and the appearance of malignant thyroid neoplasm.* The author, on the basis of the cases reported in the literature, states that radiation therapy of the neck and mediastinum is assumed to favor development of cancer in later years. His series of 12 cases of juvenile thyroid cancer includes only 1 which had received previous radiation therapy to the neck, and does not confirm this assumption.

(6) *Diagnosis of thyroid neoplasm.* The diagnosis is based principally on clinical examination and must be confirmed by biopsy. Some authors insist on the necessity of serial histologic examinations. Others control the extent of their operative intervention by frozen sections. The difficulties of the histologic diagnosis are due to the coexistence of diverse cytologic types in the same specimen, be it from a primary or a metastatic site. Roentgen examination is of great aid in establishing the diagnosis. It includes conventional roentgenography, laminagraphy, arteriography, pneumothyreography, and pneumomediastinography. It will aid in the recognition of the location and extent of the primary and secondary lesions, visceral and skeletal. The scanning of the thyroid region after the administration of I^{131} usually shows a diminished uptake at the site of malignancy. Scintiscanning of the body following proper preparation will outline metastatic foci, which have an appreciable uptake.

(7) *Therapy.* Radical surgical extirpation of the malignant thyroid, when operable, is the method of choice. It is to be followed by a period of treatment with radioiodine. Of 22 patients so treated by the author, 7 are living more than five years. In inoperable cases, in postoperative recurrences, and in metastases, the choice of external irradiation, isotope therapy, or a combination of both must be made. Each case is to be studied individually. When an appreciable uptake of radioiodine has been determined, treatment with the isotope is advisable. When the radioiodine is poorly concentrated, the primary lesion is treated by external irradiation. The metastases are then attacked by massive doses of radioiodine, administered at intervals, when they have acquired functional characteristics.

The author proposes a standardization of the classification of the various neoplastic histologic types, a universal definition of the stages of the

disease and uniformity in the dosage of radiation.—*Anthony A. Blasi, M.D.*

Barr, Nathaniel F., Stark, Mary, and Laughlin, J. S. A comparison of two fluorometers designed to measure the radiation-induced fluorescence of silver-activated glass rods. *Radiology*, Jan., 1961, *76*, 113–115. (Address: N. F. Barr, 410 E. 68th Street, New York 21, N. Y.)

The use of silver-activated phosphate glass in the form of small cylindrical rods (1×6 mm.) for dosimetry in radiobiologic, clinical, and health physics studies has led to the design of a variety of instruments for measuring the radiation-induced fluorescence. In a comparison of the results obtained with a commercially available Bausch and Lomb microdosimeter reader and a system employing Photovolt Model #540 fluorometer, it was found that the Bausch and Lomb instrument is ultimately 100 times more sensitive than the Sloan-Kettering system; however, because of the lack of uniformity (particularly in respect to the diameter) in the rods which are presently available, advantage cannot be taken of this extra sensitivity. The greater inherent sensitivity of the Bausch and Lomb reader would be of extreme importance if rods containing less silver were developed, since this would lessen their quality dependence.—*Walter H. Jarvis, Jr., M.D.*

RADIOISOTOPES

Manaro, Julio Morato, Paseyro, Pedro, and Grosso, Osvaldo F. El gammagrama y el citograma en el estudio del cancer de la glandula tiroides. (The gammagram and the cytogram in the study of thyroid carcinoma.) *An. Fac. de med. de Montevideo*, Mar.–Apr., 1960, *45*, 215–222. (From: Instituto y Clínica Endocrinológica del Ministerio de Salud Pública y Facultad de Medicina, Montevideo, Uruguay.)

Gammagrams may provide useful information in the diagnosis of thyroid tumors by showing the relationship of the thyroid gland to soft tissue masses and adjacent structures. In 8 case of thyroid gland carcinoma, 6 showed increased I^{131} uptake within forty-eight hours. This was attributed to the fact that in thyroid neoplasms seen in an early stage there was hyperplasia of that portion of the thyroid not involved by the tumor, resulting in an increased uptake of I^{131} without a rise in thyroid hormone secretion. Two cases showed uptake readings within normal limits. One of these, an adenocarcinoma, was considered to be too small to affect the thyroid function. Aspiration cytologic diagnosis was recommended as a safe simple technique which may provide valuable information of cell structure in blastomatous growths and related conditions.—*Murray M. Friedman, M.D.*

Abt, Arthur F., and Balkus, Vincent A. The radio-renogram, with radio-renografin-I^{131}, as a diagnostic aid in urologic problems. *J. Urol.*, Jan., 1961, *85*, 95–102. (From: Radioisotope and Genitourinary Services, Veterans Administration Center, Martinsburg, W. Va.)

Renography using I^{131} offers an additional test of renal function. It is particularly useful for evaluating the differential function of the two kidneys and, in conjunction with the other methods for evaluating the urinary tract, it is an addition to the armamentarium for renal function studies. This test compares favorably with conventional renal function tests, but it should not be considered to be in itself definitively diagnostic, as it can only indicate disparity in function and not exact etiologic conditions.

The authors have successfully used this test to evaluate function in patients with unilateral renal obstruction caused by calculus, tumor, or infection. It is also of some value in the detection of unilateral renal disease in association with hypertension and with the hypoplastic or absent kidney.—*George W. Chamberlin, M.D.*

Winter, Chester C., Nordyke, Robert A., and Tubis, Manuel. Clinical experience with a new test agent for the radioisotope renogram: sodium ortho-iodohippurate-I^{131} (hippuran-I^{131}). *J. Urol.*, Jan., 1961, *85*, 92–94. (From: Departments of Surgery [Division of Urology], Medicine, and Radiology, University of California Medical Center, Los Angeles, and the Radioisotope Service, Veterans Administration Center, Los Angeles, Calif.)

The authors point out that I^{131} seems to be the best available isotope to incorporate with intravenous radiopaque material for the radioisotope renogram. Diodrast I^{131} has the disadvantage of being secreted by the liver as well as the kidneys. The use of this drug sometimes necessitates large doses to block the liver excretion; furthermore, when using diodrast I^{131} it is necessary to angle the probes away from the liver, particularly on the right side. Urokon I^{131}, hypaque, or miokon I^{131} were found to be useful but their clearance from the blood is not as rapid as that of diodrast I^{131}.

Recently, Nordyke, Tubis, and their associates found that hippuran I^{131} has a much more rapid and higher secondary rise and a more rapid descent on the tracing than does diodrast or the other media. This medium is stable, heat resistant, easily manufactured, commercially available, nontoxic, and has a biologic half life of 20 minutes. Urine assays in the

normal patient reveal as high as 75 per cent recovery of hippuran I^{131} in 30 minutes. The tests can be completed in two-thirds of the time required for hypaque.

The authors recommend hippuran I^{131} for renography at the present time.—*George W. Chamberlin, M.D.*

Siurala, M., Lamberg, B.-A., Turula, K., and Harjanne, A. Studies with I^{131}-labelled fat. IV. Absorption, distribution and excretion of radioactivity after ingestion of radioactive triolein. *Scandinav. J. Clin. & Lab. Invest.*, Jan., 1960, *12*, 97–106. (From: The Medical Department, Maria Hospital and the IVth Department of Medicine, University of Helsinki, Helsinki, Finland.)

The authors have measured plasma, urine, and fecal concentrations of I^{131} following the ingestion of radioactive triolein in 8 patients, 3 of whom had steatorrhea. In the normals, after eight hours, only 11 per cent of the ingested dose was not accounted for and total recovery eventually approached 100 per cent. Results show that most of the plasma I^{131} is contained in the nonprecipitated portion (20 per cent trichloroacetic acid was used for precipitation), and that total plasma radioactivity as it represents the iodine diffusion space is of greater practical significance than the plasma lipid bound radioactivity (precipitated portion). Total plasma recovery ranged from 47 to 63 per cent in the normals at six hours, but only 14 to 32 per cent in the patients with steatorrhea. It is also suggested that measurement of the radioactivity in the urine during the first four to six days may be a useful test for determining the amount absorbed, as the per cent recovered in normals approached 100 per cent but was much less in the abnormals.—*William K. Littman, M.D.*

Lamberg, B.-A., Siurala, M., Turula, K., Hakkila, R., and Harjanne, A. Studies with I^{131}-labelled fat. V. Distribution of radioactivity after intravenous injection of radioactive triolein emulsion. *Scandinav. J. Clin. & Lab. Invest.*, Jan., 1960, *12*, 107–115. (From: The Medical Department, Maria Hospital and the IVth Department of Medicine, University of Helsinki, Helsinki, Finland.)

This report is a continuation of the authors' previous study of oral ingestion of radioactive triolein. However, this time the triolein was given intravenously and the circulating I^{131} was studied. About 97 per cent of the lipid bound radioactivity was shown to be removed from the blood in thirty minutes. This study again confirms that measurement of only the lipid bound I^{131} in plasma after oral or intravenous administration of labelled fat does not give reliable measurement of the amount absorbed or of distribution within the body. The most important fraction is the total plasma I^{131}, which is in accordance with the results of the authors' previous studies.—*William K. Littman, M.D.*

Ruffin, Julian M., and Tyor, Malcolm P. Radioisotopes as diagnostic aids in gastroenterology; an evaluation. *J.A.M.A.*, Feb. 25, 1961, *175*, 685–688. (From: The Department of Medicine, Duke University Medical Center and the Veterans Administration Hospital, Durham, N. C.)

I^{131} labeled lipids have been used in the study of intestinal absorption since 1949. At the present time, I^{131} labeled triolein and oleic acid are the most widely employed. Oleic acid is readily absorbed by the normal intestine, whereas triolein must be partially hydrolyzed before absorption can take place. Thus, by using the above materials one can differentiate a defect in digestion from an impairment in absorption.

Blood and stool samples are studied for their radioactivity and the results are considered to be normal if the average of the fourth, fifth and sixth hour blood samples is more than 10 per cent of the administered dose and if less than 5 per cent of the administered dose is present in the 48 to 72 hour stool specimens. Organic disease may exist even if the triolein studies are normal but the reverse is not true. The I^{131} labeled fat absorption studies are of value in steatorrhea, disease of the pancreas, sprue, evaluation of treatment of sprue and in differentiating diarrhea due to an irritable colon from that due to organic disease.

I^{131} labeled human serum albumin has been used to study protein absorption. However, proteins not undergoing normal digestion and absorption are decomposed by bacterial action resulting in abnormal amounts of indol and phenol derivatives in the blood and urine. I^{131} labeled polyvinylpyrrolidone has been used recently, since it does not undergo enzymatic action and, otherwise, reacts similarly to albumin. It has been used in studying idiopathic hypoproteinemia and also for evaluating protein loss in regional enteritis, ulcerative colitis and hypertrophic gastritis.

Cr^{51} labeled red blood cells have been used to study gastrointestinal bleeding. The patient's own red blood cells are tagged and injected intravenously; the stools are collected for 24 hour periods and, by comparing the radioactivity in the stools with that in the blood, the daily blood loss can be determined. The site of bleeding can also be determined by obtaining samples at different levels by means of gastrointestinal intubation. The Cr^{51} test is believed to be more specific and more sensitive than the chemical measurements currently employed.

Co^{60} labeled vitamin B_{12} has been used to study pernicious anemia, sprue and the blind loop syndrome. The absorption of Co^{60} labeled vitamin B_{12} is impaired in sprue and is not improved by the addition of either an intrinsic factor or treatment with

antibiotics. There is malabsorption of Co^{60} labeled vitamin B_{12} in pernicious anemia, which is corrected by the addition of an intrinsic factor. The absorption of Co^{60} labeled vitamin B_{12} is impaired in the blind loop syndrome (diverticulosis, blind loops, or strictures with secondary stasis and obstruction) and returns to normal following the addition of an antibiotic.

The above radioisotopes have been found to be useful in studying various aspects of digestion. Some of the tests do not correlate too closely with the chemical quantitative tests, but this does not detract from their usefulness. New uses of the present materials and the development of new materials in the study of digestion are to be anticipated.—*Charles W. Cooley, M.D.*

Cordrey, Lee J., Christie, James H., MacIntyre, William J., and Palmer, Rufus. A study of strontium 85 metabolism in the normal and in the castrated mouse. *J. Bone & Joint Surg.*, Dec., 1960, *42-A*, 1349–1362. (From: The Radioisotope Laboratory, Highland View Hospital, Cleveland, Ohio.)

The purpose of this study was to determine in mice the effect of castration on bone. Radioactive strontium 85 (Sr^{85}) was used because of its close similarity to calcium regarding its biologic behavior and especially regarding bone metabolism. Radioactive isotope evaluation was used because continuous results can be quantitated by this method. Sr^{85} was administered intraperitoneally to obviate the difference between calcium and strontium absorption from the intestinal tract.

The mice were injected with 10 μc of $Sr^{85}Cl_2$, and a similar amount was injected into a serum bottle filled with distilled water (approximately the size of a mouse). The radioactivity of each mouse was then counted (in a small cardboard box to determine the exact amount injected) and this was compared with the standard and normalized to 100 per cent; the subsequent disappearance of Sr^{85} from the mice was determined on this basis.

The effect of castration on some of the chemical values of bone was determined first, and it was shown that the calcium values in castrated mice were markedly elevated in the period immediately following castration and then fell off progressively with time; after five weeks the values were uniformly below the normal range. The biologic turnover of Sr^{85} was determined on a group of mice and it was found that there were three phases: (1) an initial relatively rapid turnover which lasted for forty-eight days, (2) a slower decrease in excretion, and (3) a very slight release of Sr^{85}. The distribution of Sr^{85} in the mouse was studied and, five days after the injection of Sr^{85}, 99 per cent was found to be retained in the skeleton. It was also determined that the younger mice excreted Sr^{85} more rapidly than the older mice.

The retention of Sr^{85} in a castrated mouse depended on the time it was injected after castration. In the group injected with Sr^{85} two weeks after castration, there was no difference between this group and the control group. There was a definite increase in retention of Sr^{85} in the group injected four weeks after castration, and this was more marked in the group injected eight weeks after castration. The amount of Sr^{85} retained in the latter group is comparable to the calcium loss. The increased retention of Sr^{85} during the period of calcium loss is believed to be due to a hetero-ionic replacement of calcium ions by the strontium ions.—*Charles W. Cooley, M.D.*

MISCELLANEOUS

Maestri, A., Marcato, M., Neri, F., and Taverna, L. Irradiazione cardiaca quale tentativo di prevenzione dell'infarto; studio arteriografico della circolazione coronarica in cani sottoposti ad infarto sperimentale previo trattamento radiante. (Irradiation of the heart as a possible preventative of infarction; arteriographic studies of the coronary circulation in dogs with experimental infarct which was preceded by radiation therapy.) *Ann. radiol. diag.*, 1960, *33*, 426–452. (From: Istituto di Clinica Chirurgica Generale e Terapia Chirurgica dell'Università di Parma and Istituto di Radiologia Medica dell'Università di Parma, Parma, Italy.)

The purpose of this experiment was to study the effects of moderate doses of radiation on the heart, followed by ligation of the descending branch of the anterior coronary artery to produce an acute coronary occlusion.

Thirty dogs were used in this experiment: 10 dogs received 1,500 r to the cardiac area; 10 received 2,000 r, and 10 were used as controls. Following irradiation of the 20 dogs, all, including the controls, were subjected to ligation of the descending branch of the anterior coronary artery. The ligature was placed at various sites, such as at the origin of the vessel, about 2 cm. distal to the origin, or at the greatest possible distance from its origin.

Recorded results concerned survival and the establishment of collateral circulation in the myocardium. Coronary angiography was used either *in vivo* or in autopsy specimens to study the vascular bed. The results show a higher percentage of survival in the irradiated group than in the controls. There was no appreciable difference between the dogs receiving 1,500 r or 2,000 r. Coronary arteriography showed that collateral and compensatory circulation developed to a greater degree in the irradiated myocardium.

The authors conclude that the myocardium which

has received a moderate dose of radiation can better cope with an acute coronary occlusion, because of its increased ability to establish collateral circulation, than the nonirradiated myocardium.

This is a very interesting and thought-provoking article. The illustrations are numerous and of excellent quality but perhaps could be more convincing. The authors are to be complimented for their excellent handling of the details of such a delicate experiment.—*Peter E. Russo, M.D.*

Maestri, A., and Dazzi, C. Ricerche sperimentali microradiografiche sulla vascolarizzazione del miocardio in condizioni di normalità e dopo trattamento radiante. (Experimental microradiographic research on the vascularity of the myocardium of the normal heart and following radiation therapy.) *Ann. radiol. diag.*, 1960, *33*, 453–470. (From: Istituto di Radiologia dell'Università di Parma, Parma, Italy.)

The authors state that previous experiments concerning the heart and coronary circulation showed that moderate doses of radiation produce hyperemia and favorably improve the vascularity of the organ. The results were evaluated arteriographically. In the present experiment, microangiographic methods were employed to study these changes in the vascular supply of the myocardium.

A group of 20 dogs was used. A dose of 2,000 r, with conventional treatment factors, was given over the cardiac area. In 10 dogs the dose was protected over a period of twenty days; the other 10 dogs were used as controls. About a week after irradiation was completed, all of the dogs were sacrificed.

A solution of barium sulfate was used to inject the coronary vessels of the heart. The pressure applied to this injection was sufficient to fill the most terminal capillaries. In none of the specimens was contrast medium found in the venous side of the circulation. After the injection the coronary arteries were ligated at their origin. In some of the specimens, prior to the injection of the opaque material, the descending branch of the anterior coronary artery was ligated in an effort to demonstrate, if possible, the collateral circulation to this occluded vessel. The injected hearts were kept at temperatures below 0° to solidify the contrast medium and to prevent compression of the smaller vessels during the preparation of the microscopic sections. The preparations were then fixed in 10 per cent formalin. Microradiographs of these specimens were studied for vascularity of the myocardium.

The authors arrived at the following conclusions: Irradiation of the myocardium with a certain dose produces vasodilatation of the coronary arteries, especially at the precapillary level. Also the collateral and communicating branches become more functional. These terminal collateral and communicating branches vary from 20 to 200 μ in width and 2 to 3 cm. in length; they can also be demonstrated in the myocardia which did not receive irradiation but are smaller in caliber and less numerous in number. Following ligation of the descending branch of the anterior coronary artery, anastomosis with adjacent vessels in the affected area can be shown. These collateral and communicating branches are not newly formed vessels but are present in a latent state.

The article contains many illustrations of microradiographs which appear to be rather convincing. It is, however, disturbing to know that irradiation, in moderate doses, is used to control vascularization in tissues such as the cornea and conjunctiva.—*Peter E. Russo, M.D.*

Levy, Marie L., Palazzi, Hugo M., Nardi, George L., and Bunker, John P. Hepatic blood flow variations during surgical anesthesia in man measured by radioactive colloid. *Surg., Gynec. & Obst.*, Mar., 1961, *112*, 289–294. (From: The Anesthesia Laboratory and the Department of Surgery of the Harvard Medical School at the Massachusetts General Hospital, Boston, Mass.)

Hepatic blood flow was studied in 68 patients undergoing a variety of surgical procedures during thiopental, cyclopropane, ether, local and spinal anesthesia.

Hepatic plasma flow was determined by measuring the clearance of injected radioactive colloid chromic phosphate (P^{32}) by the reticuloendothelial system of the liver.

The following conclusions were reached: premedication with pentobarbital and atropine produces only slight changes. There is depression in liver blood flow during general and spinal anesthesia. Changes in liver blood flow reflect changes in arterial pressure, in portal venous pressure and in vascular resistance within the liver. During thiopental and cyclopropane anesthesia, changes in hepatic plasma flow were consistent and significant. During ether anesthesia, changes were variable and of small magnitude. This is explained by the release of epinephrine and norepinephrine into the blood with ether as an anesthetic agent. At the end of anesthesia, the difference between various agents disappears and the final mean hepatic blood flow is very nearly the same for all agents. Cirrhotic and noncirrhotic livers respond similarly.—*Bernard Loitman, M.D.*